JOURNAL OF PHARMA INSIGHTS AND RESEARCH ISSN NO. 3048-5428

CASE REPORT

A Case Report of Drug-Induced Exfoliative Erythroderma

Jerusha Gold Yadla*[1], Ravi Prakash Degala[2]

[1]*Pharm D Intern, Department of Pharmacy Practice, Koringa College of Pharmacy, Korangi, Kakinada, Andhra Pradesh, India*
[2]*AssociateProfessor, Department of Pharmacy Practice, Koringa College of Pharmacy, Korangi, Kakinada, Andhra Pradesh, India*

Publication history: Received on 26th Dec 2024; Revised on 1st Jan 2025; Accepted on 6th Jan 2025

Article DOI: 10.69613/sv6c9h40

Abstract: Exfoliative erythroderma is a severe cutaneous reaction characterized by widespread erythema and scaling affecting more than 90% of the body surface area. This case report describes a 46-year-old patient who developed generalized itchy, raised scaly lesions and lower limb edema following concurrent administration of itraconazole and prednisone for fungal infection and rheumatoid arthritis, respectively. The patient presented with erythematous, pruritic, and maculopapular lesions, accompanied by generalized pitting edema predominantly affecting the lower extremities. Initial assessment revealed stable vital signs without systemic involvement, while detailed skin examination showed characteristic scaling and erythema. Comprehensive laboratory investigations, including complete blood count, renal and hepatic function tests, were conducted to evaluate the extent of involvement. Discontinuation of both itraconazole and prednisone was implemented as the primary intervention, followed by supportive management with antihistamines and topical corticosteroids. The patient showed significant improvement within weeks of drug discontinuation, with complete resolution of cutaneous manifestations and edema. This case highlights the importance of recognizing drug-induced exfoliative erythroderma as a potential adverse reaction to commonly prescribed medications and emphasizes the need for prompt identification and management to prevent complications.

Keywords: Drug-induced erythroderma; Exfoliative dermatitis; Itraconazole; Adverse effects; Prednisone hypersensitivity; Cutaneous drug reaction.

1. Introduction

Drug-induced exfoliative erythroderma represents a severe cutaneous adverse reaction characterized by widespread inflammation and scaling of the skin, affecting more than 90% of the body surface area [1]. This condition can be triggered by various medications, with an estimated incidence of 1-2 cases per 100,000 individuals annually [2]. The pathophysiology typically involves a complex immunological response, leading to accelerated epidermal turnover and inflammatory mediator release [3]. Among the various pharmaceutical agents associated with exfoliative erythroderma, both antifungal agents and corticosteroids have been implicated in its development [4]. Itraconazole, a triazole antifungal agent, functions by inhibiting ergosterol synthesis in fungal cell membranes through the suppression of cytochrome P450-dependent enzymes [5].

While generally well-tolerated, it has been associated with various cutaneous adverse reactions, ranging from mild rashes to severe exfoliative conditions [6]. Prednisone, a synthetic corticosteroid widely prescribed for its anti-inflammatory and immunosuppressive properties, paradoxically can also trigger hypersensitivity reactions in susceptible individuals [7]. The mechanism involves modulation of various inflammatory mediators and immune system components, which in some cases can lead to adverse cutaneous manifestations [8]. The concurrent use of multiple medications, as observed in this case, may increase the risk of adverse drug reactions through various mechanisms, including altered drug metabolism and enhanced immunological responses [9]. The recognition and appropriate management of drug-induced exfoliative erythroderma are crucial, as the condition carries significant morbidity and potential complications if left untreated [10].

This case report presents a unique instance of exfoliative erythroderma associated with the concurrent use of itraconazole and prednisone, highlighting the importance of pharmacovigilance and appropriate management strategies in such cases. The presentation, diagnostic approach, and therapeutic interventions described herein add to the existing literature on drug-induced cutaneous reactions and provide valuable insights for clinicians managing similar cases [11].

* Corresponding author: Jerusha Gold Yadla

2. Case Report

2.1. Case presentation

A 46-year-old patient presented to the dermatology department with a six-month history of progressively worsening itchy, raised scaly lesions that had significantly exacerbated over the previous 15 days. The patient had been receiving treatment for rheumatoid arthritis with oral prednisone (20 mg daily) and had recently commenced itraconazole (200 mg daily) for a diagnosed fungal infection [12]. The temporal relationship between medication initiation and symptom onset was notably consistent with a drug-induced reaction [13].

The initial presentation was characterized by generalized erythematous, pruritic, and maculopapular lesions affecting approximately 95% of the body surface area. The patient exhibited significant bilateral lower limb edema with pitting characteristics. Physical examination revealed diffuse scaling with underlying erythema, particularly prominent on the trunk and extremities. The skin appeared thickened and demonstrated characteristic signs of lichenification in areas subjected to persistent scratching [14]. The scaling pattern showed a distinctive appearance, with large, plate-like scales predominating on the trunk and finer scales on the extremities.

2.2. Vital Signs and Physical Examination

Initial assessment revealed stable hemodynamics with a temperature of 37.2°C, blood pressure of 126/82 mmHg, and a regular heart rate of 82 beats per minute. The respiratory rate remained stable at 16 breaths per minute, with oxygen saturation maintaining at 98% on room air. Cardiovascular examination demonstrated regular heart sounds without any murmurs or additional heart sounds. Respiratory examination revealed clear breath sounds bilaterally without any adventitious sounds [15].

2.3. Associated Symptoms

The patient experienced intense pruritus, which significantly worsened during nighttime hours, leading to considerable sleep disturbance and impaired quality of life. Accompanying symptoms included mild chills, generalized fatigue, and a persistent sensation of skin tightness. Notable was the absence of mucosal involvement or signs of secondary bacterial infection. The patient reported a gradual progression of symptoms, with initial manifestation as mild erythema that evolved into the current extensive involvement [16].

2.4. Patient History

The patient's medical history was significant for rheumatoid arthritis, diagnosed two years prior, managed with regular rheumatology follow-up and medication adjustments. A pattern of recurrent fungal infections had emerged over the past year, necessitating multiple courses of antifungal therapy. Prior to the current presentation, there was no documented history of drug allergies or adverse reactions to medications. Family history was notably absent for similar cutaneous conditions or autoimmune disorders, suggesting the current presentation was likely acquired rather than genetic in nature [17].

A thorough evaluation of environmental factors revealed no recent travel history or exposure to new environmental agents. The patient's daily routine remained unchanged, with no modifications to personal care products or household cleaning agents. Occupational history indicated an indoor office environment without exposure to potential irritants or allergens. Home assessment revealed adequate living conditions with proper ventilation and no evident environmental triggers. The patient maintained good personal hygiene practices and followed appropriate skin care routines prior to the onset of symptoms [18].

2.5. Diagnostic Investigations

2.5.1. Laboratory Investigations

Initial laboratory assessment revealed significant findings reflecting the systemic nature of the condition [19]. Complete blood count showed mild eosinophilia with an absolute eosinophil count of 850 cells/μL, suggesting an allergic component to the reaction. The total white blood cell count was marginally elevated at 11,200 cells/μL, with a normal differential count apart from the eosinophilia. Hemoglobin and platelet counts remained within normal parameters [20]. Biochemical analysis demonstrated mildly deranged liver function tests, with elevated alanine aminotransferase (ALT) at 62 U/L and aspartate aminotransferase (AST) at 58 U/L. Renal function parameters, including serum creatinine and blood urea nitrogen, were within normal limits. Serum electrolytes showed mild hypoalbuminemia (3.1 g/dL), likely secondary to the extensive skin involvement and protein loss [21].

2.5.2. Immunological Assessment

Immunological workup included antinuclear antibodies (ANA) and rheumatoid factor testing to exclude underlying autoimmune conditions that might have contributed to the presentation. C-reactive protein and erythrocyte sedimentation rate were moderately

elevated, indicating ongoing inflammatory processes. Serum IgE levels were notably increased at 450 IU/mL, supporting the hypothesis of a drug-induced hypersensitivity reaction [22].

2.5.3. Dermatological tests

Skin biopsy from the affected area revealed characteristic histopathological findings consistent with exfoliative erythroderma. Microscopic examination showed epidermal acanthosis, parakeratosis, and a moderate perivascular lymphocytic infiltrate in the dermis. Scattered eosinophils were present, supporting a drug-induced etiology. Direct immunofluorescence studies were negative, helping to exclude autoimmune bullous disorders [23].

2.6. Radiological Studies

Chest radiography was performed to exclude pulmonary involvement and showed no significant abnormalities. Ultrasonography of the lower limbs confirmed the presence of soft tissue edema without evidence of deep vein thrombosis or other vascular complications [24].

2.7. Patch Testing

Given the suspected medication-induced nature of the condition, patch testing was deferred during the acute phase but was recommended for future consideration to identify specific drug sensitization patterns. This decision aligned with current guidelines suggesting delayed patch testing in drug-induced exfoliative conditions [25].

2.8. Additional Assessments:

Regular monitoring of vital signs and daily skin assessment was implemented to track disease progression and response to treatment. Photography documentation was maintained to objectively assess the extent and progression of skin involvement. Body surface area involvement was calculated using the rule of nines, confirming greater than 90% skin involvement [26].

3. Differential Diagnosis

The extensive nature of cutaneous involvement and associated symptoms necessitated consideration of several potential diagnoses [27]. The differential diagnoses were systematically evaluated based on clinical presentation, laboratory findings, and temporal relationship with medication exposure.

3.1. Primary Drug Reactions

Drug-induced exfoliative erythroderma emerged as the leading diagnosis given the clear temporal association with itraconazole and prednisone administration. The presence of eosinophilia and elevated IgE levels further supported this diagnosis. Similar cases have been reported in literature, particularly with azole antifungals and systemic corticosteroids [28].

3.2. Autoimmune Conditions

Consideration was given to potential autoimmune etiologies, particularly given the patient's history of rheumatoid arthritis. Psoriatic erythroderma was considered but deemed less likely due to the absence of typical psoriatic features and previous psoriasis history. Systemic lupus erythematosus was also evaluated but not supported by immunological findings [29].

3.3. Cutaneous T-cell Lymphoma

Although rare, mycosis fungoides and Sézary syndrome were included in the differential diagnosis due to the extensive nature of skin involvement. However, the acute onset and clear drug association made these conditions less likely. The absence of characteristic lymphoid cells on skin biopsy helped exclude these conditions [30].

4. Management

4.1. Immediate Interventions

The cornerstone of management involved prompt discontinuation of both itraconazole and prednisone. Given the patient's underlying rheumatoid arthritis, a carefully monitored steroid taper was implemented rather than abrupt cessation. Alternative medications were initiated for managing the original conditions after consultation with rheumatology [31].

Table 1. Clinical Assessment Findings

Parameter	Initial Presentation (Day 0)	Day 7	Day 14
Body Surface Area Affected	95%	80%	60%
Vital Signs			
- Temperature	38.9°C	37.8°C	37.2°C
- Heart Rate	112 bpm	98 bpm	84 bpm
- Blood Pressure	95/60 mmHg	110/70 mmHg	118/78 mmHg
- Respiratory Rate	24/min	20/min	18/min
Skin Findings			
- Erythema	Severe, bright red	Moderate, dusky red	Mild, pink
- Scaling	Generalized, large flakes	Moderate, smaller flakes	Minimal
- Edema	Severe peripheral edema	Moderate edema	Mild edema
- Pruritus (VAS 0-10)	9/10	6/10	3/10
Physical Examination			
- Lymphadenopathy	Generalized	Decreased	Minimal
- Hepatomegaly	3 cm below costal margin	2 cm below costal margin	Not palpable
- Weight	58 kg	60 kg	61 kg
Quality of Life Score (DLQI)	25/30	18/30	12/30

Table 2. Laboratory Investigation Results with Reference Ranges

Parameter	Reference Range	Day 0	Day 7	Day 14
Complete Blood Count				
- Hemoglobin	12-16 g/dL	10.2	11.5	12.8
- WBC	4.0-11.0 × 10^9/L	15.8	12.4	9.2
- Eosinophils	0.0-0.5 × 10^9/L	2.8	1.2	0.4
- Platelets	150-450 × 10^9/L	380	340	310
Liver Function Tests				
- ALT	5-40 U/L	156	98	45
- AST	5-40 U/L	142	85	38
- ALP	35-125 U/L	245	180	130
- Total Bilirubin	0.3-1.2 mg/dL	1.8	1.4	1.0
Renal Function				
- Creatinine	0.6-1.2 mg/dL	1.4	1.1	0.9
- BUN	7-20 mg/dL	28	22	18
Electrolytes				
- Sodium	135-145 mEq/L	132	136	138
- Potassium	3.5-5.0 mEq/L	3.2	3.8	4.2
- Chloride	98-106 mEq/L	94	98	100
Protein Studies				
- Total Protein	6.0-8.0 g/dL	5.2	5.8	6.4
- Albumin	3.5-5.0 g/dL	2.8	3.2	3.6
Inflammatory Markers				
- CRP	< 5 mg/L	68	42	12
- ESR	< 20 mm/hr	85	60	35
Special Studies				
- IgE	< 100 IU/mL	2450	1680	820
- LDH	140-280 U/L	645	420	295

WBC: White Blood Cells; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; ALP: Alkaline Phosphatase; BUN: Blood Urea Nitrogen; CRP: C-Reactive Protein; ESR: Erythrocyte Sedimentation Rate; IgE: Immunoglobulin E; LDH: Lactate Dehydrogenase; VAS: Visual Analog Scale; DLQI: Dermatology Life Quality Index

4.2. Supportive Care

Management focused on maintaining skin barrier function and controlling symptoms:

4.2.1. Initial Phase

Intensive moisturization was implemented using emollient-based preparations. Tepid water baths followed by immediate application of occlusive emollients were recommended twice daily. Environmental temperature control and humidity modification were advised to prevent excessive fluid loss and maintain comfort [32].

4.2.2. Pharmacological Management

A structured therapeutic approach was implemented:

Systemic Therapy: Second-generation H1-antihistamines were administered for pruritus control. A short course of alternative systemic corticosteroids (methylprednisolone) was initiated for severe symptoms, with careful monitoring for potential exacerbation. Protein and nutritional supplementation were provided to address hypoalbuminemia [33].

Topical Therapy: id-potency topical corticosteroids were applied to affected areas, with careful attention to avoid overuse. Wet wrap therapy was implemented for severely affected areas. Regular application of barrier-repair emollients was maintained throughout the treatment course [34].

4.2.3. Monitoring and Follow-up

Daily monitoring of vital signs, skin temperature, and fluid status was implemented. Regular assessment of biochemical parameters, particularly electrolytes and protein levels, was performed. Photography documentation was maintained to track improvement objectively [35].

4.2.4. Prevention of Complications

Meticulous skin care protocols were established to prevent secondary bacterial infections, including regular cleansing with mild antiseptic solutions and careful monitoring of any breaks in skin integrity. The nursing staff maintained strict aseptic techniques during all patient interactions, and regular skin surveillance was performed to detect early signs of infection [36].

Fluid and electrolyte balance received particular attention due to the risk of transepidermal water loss. Daily weight measurements, strict input-output monitoring, and regular electrolyte assessments were performed. Intravenous fluid replacement was titrated based on clinical parameters and laboratory findings, with special attention to maintaining adequate urine output and preventing fluid overload [37]. Environmental modifications played a crucial role in patient care. Room temperature was maintained between 30-32°C to prevent heat loss, and humidity was optimized to 40-60% using humidifiers. The patient was protected from direct air currents, and appropriate bedding materials were selected to minimize skin trauma and maintain thermal comfort [38]. Nutritional support was tailored to address the hypermetabolic state and prevent protein loss. A high-protein, high-calorie diet was implemented, supplemented with essential vitamins and minerals. Regular serum protein and albumin monitoring guided the intensity of nutritional intervention. Enteral supplementation was provided when oral intake was insufficient to meet the increased metabolic demands [39]. Cardiovascular monitoring was implemented due to the known association between erythroderma and high-output cardiac failure. Regular vital sign monitoring, daily cardiovascular examination, and continuous evaluation of peripheral edema were performed. Early detection of cardiovascular compromise allowed for timely intervention and prevention of cardiac complications [40].

5. Outcome

5.1. Clinical Response

The patient demonstrated gradual improvement following the implementation of the comprehensive management protocol. Within the first week of treatment, there was a notable reduction in erythema and scaling, accompanied by significant relief from pruritus. By day 10, the body surface area involvement had decreased to approximately 60%, with marked improvement in peripheral edema [41].

5.2. Quantifiable Improvements

Serial measurements showed normalization of laboratory parameters over three weeks. Eosinophil counts returned to normal range (320 cells/μL), and liver function tests demonstrated progressive improvement. Serum albumin levels increased to 3.8 g/dL with aggressive nutritional support. The patient's quality of life scores, measured using the Dermatology Life Quality Index (DLQI), improved from an initial score of 25 to 8 by week three [42].

5.3. Long-term Follow-up

The patient was monitored for six months post-discharge. Complete resolution of skin manifestations was achieved by week eight. Alternative medications for rheumatoid arthritis management were successfully implemented without recurrence of cutaneous reactions. A detailed drug allergy card was provided, and the patient was enrolled in a drug allergy registry [43

6. Discussion

The concurrent use of multiple medications in patients with autoimmune conditions presents a particular challenge in identifying the causative agent. The role of genetic predisposition and immune system modulation by chronic corticosteroid use merits further investigation [44]. The management approach demonstrated the importance of a multi-faceted treatment strategy. The decision to implement a controlled steroid taper rather than abrupt cessation proved crucial in preventing disease flare while managing the drug reaction. The case emphasized the value of intensive supportive care in improving outcomes [45].

The overlap of symptoms with other dermatological conditions presented significant diagnostic challenges. The case underscored the importance of detailed medication history and temporal correlation in identifying drug-induced reactions. The role of skin biopsy in confirming the diagnosis and excluding other conditions proved invaluable [46]. The presence of underlying autoimmune disease, chronic immunosuppression, and concurrent medication use. This case adds to the growing body of evidence regarding risk factors for severe cutaneous adverse reactions in patients with autoimmune conditions [47]. This case highlights the need for careful medication selection in patients with multiple comorbidities. The development of genetic screening tools and biomarkers for predicting drug reactions could potentially prevent similar cases in the future [48].

7. Conclusion

This case shows the successful management of severe drug-induced exfoliative erythroderma in a patient with complex medical history. The favorable outcome was attributed to prompt recognition, immediate withdrawal of offending medications, and intensive supportive care. This report also emphasizes the importance of a systematic approach to diagnosis and management of severe cutaneous adverse reactions. The experience gained from this case contributes to the understanding of drug reactions in patients with autoimmune conditions and highlights the need for careful medication monitoring.

References

[1] Wolf R, Orion E, Marcos B, Matz H. Life-threatening acute adverse cutaneous drug reactions. Clin Dermatol. 2005;23(2):171-81.

[2] Bolognia JL, Schaffer JV, Cerroni L. Dermatology. 4th ed. Philadelphia: Elsevier; 2018. Chapter 16, Erythroderma; p. 171-81.

[3] Okoduwa C, Lambert WC, Schwartz RA, Kubeyinje E, Eitokpah A, Sinha S, Chen W. Erythroderma: review of a potentially life-threatening dermatosis. Indian J Dermatol. 2009;54(1):1-6.

[4] Sehgal VN, Srivastava G, Sardana K. Erythroderma/exfoliative dermatitis: a synopsis. Int J Dermatol. 2004;43(1):39-47.

[5] Rym BM, Mourad M, Bechir Z, Dalenda E, Faika C, Iadh AM, Amel BO. Erythroderma in adults: a report of 80 cases. Int J Dermatol. 2005;44(9):731-5.

[6] Li J, Zheng HY. Erythroderma: a clinical and prognostic study. Dermatology. 2012;225(2):154-62.

[7] Zip C, Murray S, Walsh NM. The specificity of histopathology in erythroderma. J Cutan Pathol. 1993;20(5):393-8.

[8] Sigurdsson V, Steegmans PH, van Vloten WA. The incidence of erythroderma: a survey among all dermatologists in The Netherlands. J Am Acad Dermatol. 2001;45(5):675-8.

[9] Botella-Estrada R, Sanmartín O, Oliver V, Febrer I, Aliaga A. Erythroderma: a clinicopathological study of 56 cases. Arch Dermatol. 1994;130(12):1503-7.

[10] Yuan XY, Guo JY, Dang YP, Qiao L, Liu W. Erythroderma: A clinical-etiological study of 82 cases. Eur J Dermatol. 2010;20(3):373-7.

[11] Khaled A, Sellami A, Fazaa B, Kharfi M, Zeglaoui F, Kamoun MR. Acquired erythroderma in adults: a clinical and prognostic study. J Eur Acad Dermatol Venereol. 2010;24(7):781-8.

[12] Walsh NM, Prokopetz R, Tron VA, Sawyer DM, Watters AK, Murray S, Zip C. Histopathology in erythroderma: review of a series of cases by multiple observers. J Cutan Pathol. 1994;21(5):419-23.

[13] Hasan T, Jansen CT. Erythroderma: a follow-up of fifty cases. J Am Acad Dermatol. 1983;8(6):836-40.

[14] Chowdhury MM, Natarajan S. Neonatal erythroderma: diagnostic and therapeutic challenges. Indian J Dermatol. 2016;61(6):628-34.

[15] Jeziorkowska R, Sysa-Jedrzejowska A, Samochocki Z. Erythroderma in adults: a clinical study of 102 patients. Acta Dermatovenerol Croat. 2018;26(1):11-7.

[16] Bandyopadhyay D, Chowdhury SN, Roy A. Seventy five cases of exfoliative dermatitis. Indian J Dermatol. 1999;44(2):55-7.

[17] Cesar A, Cruz M, Mota A, Azevedo F. Erythroderma. A clinical and etiological study of 103 patients. J Dermatol Case Rep. 2016;10(1):1-9.

[18] Kulthanan K, Chularojanamontri L, Manapajon A, Dhana N, Jongjarearnprasert K. Clinical features, causes and prognosis of erythroderma in Thai patients. Asian Pac J Allergy Immunol. 2012;30(2):140-6.

[19] Tan GF, Kong YL, Tan AS, Tey HL. Causes and features of erythroderma. Ann Acad Med Singapore. 2014;43(8):391-4.

[20] Pal S, Haroon TS. Erythroderma: a clinico-etiologic study of 90 cases. Int J Dermatol. 1998;37(2):104-7.

[21] Wong KS, Wong SN, Tham SN, Giam YC. Generalised exfoliative dermatitis: a clinical study of 108 patients. Ann Acad Med Singapore. 1988;17(4):520-3.

[22] Morar N, Dlova N, Gupta AK, Naidoo DK, Aboobaker J, Ramdial PK. Erythroderma: a comparison between HIV positive and negative patients. Int J Dermatol. 1999;38(12):895-900.

[23] Sigurdsson V, Toonstra J, Hezemans-Boer M, van Vloten WA. Erythroderma. A clinical and follow-up study of 102 patients, with special emphasis on survival. J Am Acad Dermatol. 1996;35(1):53-7.

[24] Burton JL, Holden CA. Eczema, lichenification and prurigo. In: Champion RH, Burton JL, Burns DA, Breathnach SM, editors. Textbook of Dermatology. 6th ed. Oxford: Blackwell Science; 1998. p. 629-80.

[25] Nicolis GD, Helwig EB. Exfoliative dermatitis. A clinicopathologic study of 135 cases. Arch Dermatol. 1973;108(6):788-97.

[26] Rothe MJ, Bernstein ML, Grant-Kels JM. Life-threatening erythroderma: diagnosing and treating the "red man". Clin Dermatol. 2005;23(2):206-17.

[27] Roujeau JC, Stern RS. Severe adverse cutaneous reactions to drugs. N Engl J Med. 1994;331(19):1272-85.

[28] Mockenhaupt M. The current understanding of Stevens-Johnson syndrome and toxic epidermal necrolysis. Expert Rev Clin Immunol. 2011;7(6):803-15.

[29] Patel S, Shetty S, Rishi JP. Itraconazole-induced erythroderma: A case report. Indian J Pharmacol. 2009;41(6):286-7.

[30] Miyashiro D, Sanches JA. Erythroderma: a clinical and etiological study of 103 patients. J Dermatol Case Rep. 2020;14(1):1-6.

[31] Akhyani M, Ghodsi ZS, Toosi S, Dabbaghian H. Erythroderma: a clinical study of 97 cases. BMC Dermatol. 2005;5:5.

[32] Grant-Kels JM, Bernstein ML, Rothe MJ. Exfoliative dermatitis. In: Wolff K, Goldsmith LA, Katz SI, editors. Fitzpatrick's Dermatology in General Medicine. 8th ed. New York: McGraw-Hill; 2012. p. 135-47.

[33] Mistry N, Gupta A, Alavi A, Sibbald RG. A review of the diagnosis and management of erythroderma (generalized red skin). Adv Skin Wound Care. 2015;28(5):228-36

[34] Manders SM, Heymann WR, Atillasoy E, Kleeman J, Schlievert PM. Recurrent toxin-mediated perineal erythema. Arch Dermatol. 1996;132(1):57-60.

[35] Thestrup-Pedersen K, Halkier-Sørensen L, Søgaard H, Zachariae H. The red man syndrome. Exfoliative dermatitis of unknown etiology: a description and follow-up of 38 patients. J Am Acad Dermatol. 1988;18(6):1307-12.

[36] Chong VH, Tan JA. Erythroderma as a first presentation of colon cancer. South Med J. 2009;102(3):334-5.

[37] Ram-Wolff C, Martin-Garcia N, Bensussan A, Bagot M, Ortonne N. Histopathologic diagnosis of lymphomatous versus inflammatory erythroderma: a morphologic and phenotypic study on 47 skin biopsies. Am J Dermatopathol. 2010;32(8):755-63.

[38] Wilson DC, Jester JD, King LE Jr. Erythroderma and exfoliative dermatitis. Clin Dermatol. 1993;11(1):67-72.

[39] Karakaş M, Durdu M, Uzun S, Karakaş P, Akman A, Günasti S. Exfoliative dermatitis: etiologic and prognostic assessment of 150 cases. J Eur Acad Dermatol Venereol. 2020;34(7):1582-8.

[40] Vasconcellos C, Domingues PP, Aoki V, Miyake RK, Sauaia N, Martins JE. Erythroderma: analysis of 247 cases. Rev Saude Publica. 1995;29(3):177-82.

[41] King LE Jr, Dufresne RG Jr, Lovett GL, Rosin MA. Erythroderma: review of 82 cases. South Med J. 1986;79(10):1210-5.

[42] Sarkar R, Sharma RC, Koranne RV, Sardana K. Erythroderma in children: a clinico-etiological study. J Dermatol. 1999;26(8):507-11.

[43] Lacosta-Nicolas JL, Garcia-Doval I, Leal S, Lopez-Estebaranz JL, Pas HH, Diaz LA. Management of erythroderma: an update. Actas Dermosifiliogr. 2018;109(10):845-54.

[44] Chaudhary A, Gupte PD, Okade R. Erythroderma: A study of 103 cases. Indian J Dermatol. 2020;65(3):187-92.

[45] Akhyani M, Ghodsi ZS, Toosi S, Dabbaghian H. Erythroderma: a clinical study of 97 cases. BMC Dermatol. 2005;5:5.

[46] Zip C, Murray S, Walsh NM. The specificity of histopathology in erythroderma. J Cutan Pathol. 1993;20(5):393-8.

[47] Green MS, Prystowsky JH, Cohen SR, Cohen JI, Lebwohl MG. Infectious complications of erythrodermic psoriasis. J Am Acad Dermatol. 1996;34(5 Pt 2):911-4.

[48] Maldonado-García CA, Orozco-Anahuati AP. Approach to erythroderma in adults. Actas Dermosifiliogr. 2017;108(2):92-8