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Lupus-Lyell: A Rare Lupus Skin Manifestation

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Abstract **Case Report**

Introduction: Lupus-Lyell (LL) or lupus epidermal necrolysis is a rare and severe cutaneous clinical form of systemic lupus. ***Case Report:*** This article describes the case of a 22-year-old female patient with multiple skin ulcers who was diagnosed with Lupus-Lyell (Lyell-like syndrome). The article also describes the literature review on the diagnosis and treatment of this complex form of lupus disease. ***Conclusion:*** LL is a serious pathology whose diagnosis and management are complex, multidisciplinary and associated with therapeutic education.

Keywords: Lupus-Lyell – Dysimmunity - Internal Medicine.

INTRODUCTION

Systemic lupus (SL) is a non-organ-specific systemic autoimmune disease characterized by the presence of autoantibodies that cause tissue damage in multiple organs [1]. The skin is the second most commonly affected organ by SLL [1]. Gilliam and Sontheimer proposed a classification of cutaneous systemic lupus (CSL) into lupus-specific CSL (acute, subacute, and chronic) and non-lupus-specific CSL based on the presence of histologic interface dermatitis [2]. Toxic epidermal necrolysis (TEN) is a lifethreatening condition resulting in an acute erythematous blistering eruption [2]. The majority of cases appear to be related to idiosyncratic drug reactions [3]. However, in some cases, it has also been reported in association with acute graft versus host disease (GVHD), infection, vaccinations and SLE. TEN is thought to be more prevalent in SL [3].

Lupus-Lyell (LL) or toxic epidermal necrolysis lupus is an exceptional entity that can mimic in every way an authentic drug-induced Lyell syndrome [1]. It results in the sudden destruction of the superficial layer of the skin and mucous membranes. It is a hyperacute and exceptional cutaneous expression of lupus disease whose cutaneous manifestations are very polymorphic. Several clinical forms have been described but rare cases published in sub-Saharan Africa and specifically in Mali. Management in our context is based on immunomodulators, immunosuppressants associated with corticosteroid therapy. Difficulties in accessing specialized care delay diagnosis and adequate management. The prognosis is poor with a mortality rate of; the problem is to distinguish between an authentic severe drug eruption and a hyperacute expression of lupus disease. We thus describe the case of a young 22-year-old patient suffering from a severe subacute cutaneous lupus flare in the form of Lupus-Lyell in a context of familial lupus; the difficulties of specific management in this case.

OBSERVATION

We report the case of a 22-year-old patient, of Malian nationality; student. She was received on

September 9, 2024 in the Internal Medicine department of the Point G University Hospital Center in Bamako for multiple skin ulcerations.

The onset of symptoms dates back to around 6 months, marked by the spontaneous and progressive appearance of multiple ulcero-necrotic, oozing, pruritic wounds of varying sizes, evolving locally at the level of the folds (neck, submammary, between the thighs, elbow, popliteal fossa); sparing the oral and genital mucous membranes, leaving scar lesions. These lesions are associated with diffuse pain rated at 8/10 according to the simple verbal scale, without notion of medication intake. This picture would evolve with the addition of fatigue, functional impotence of the 4 limbs predominating at the level of the roots of the limbs; without notion of initial trauma, associated with a notion of dysphagia and odynophagia; and for which no therapy had been undertaken. The current episode dates back about a month, marked by the progressive installation of a painless, soft, symmetrical swelling of the lower limbs, no imprint on digital pressure, associated with permanent puffiness of the face without dyspnea. All this would evolve in a context of unquantified fever, physical asthenia, non-selective anorexia. It is in front of this symptomatology that the family consults in a clinic where the diagnosis of lupus was made, and the family consults us for management. As a history; she has had systemic lupus for 4 months in irregular care under prednisolone 20mg per day, and azathioprine 50mg per day, acute iatrogenic adrenal insufficiency under hydrocortisone 30mg per day. The patient reports that she was diagnosed with eye disorders: unspecified maculopathy at the Tropical Ophthalmology Institute of Africa in Bamako. She has an aunt with lupus; the drug investigation reveals the recent intake of spironolactone 75mg per day. She drinks tea and her diet is mainly based on cereals.

The general condition examination showed a conscious patient, bedridden, not autonomous, athletic and febrile to the touch; her Karnofsky index was 30%. The blood pressure in the sitting position in the left arm was 159/79 mmHg; a heart rate of 136 bpm; a respiratory rate of 20 cycles per minute; a left axillary temperature of 40.8°C; a capillary blood glucose level at entry of 0.97g/L. Her measurements at entry were a weight of 60kg, height of 169cm for a body mass index of 24.51kg/m² and an SpO2 of 97%.

The dermatological examination noted: Multiple ulcerations of variable size, oozing, non-hemorrhagic, with a clean whitish base, with irregular borders, painful, disseminated over the entire integument outside the anterior abdominal region and the scalp, with spaces of healthy skin; associated with post-ulceration erosions, pigmented scars in places, areas of detached and detachable skin (Image 1) ; estimated according to Wallace at 58.5% (the head 4.5% - the front thorax 9% - the right upper limb 9% the left upper limb 9% - the back 18% - the left and right lower limbs 9 and 9%). A puffy face with palpebral occlusion. The remainder of the physical examination revealed palmoplantar conjunctival pallor, proximal and bilateral motor deficit with muscle strength at 3/5 in the left upper limb versus 2/5 in the right according to the medical research council, amyotrophy of both upper limbs, slight bilateral swelling of both feet not pitting. Vaginal examination revealed thick, brittle and fetid brown leukorrhea; without associated pruritus, metrorrhagia or pelvic pain. The other devices were carefully examined and did not reveal any abnormalities.

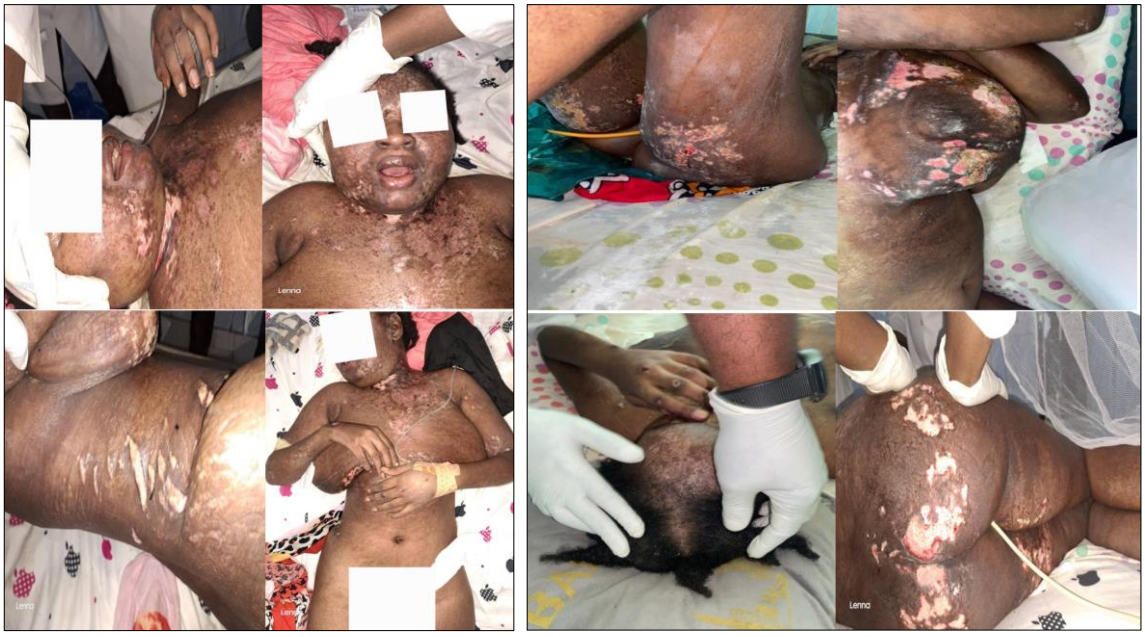

Image 1: Pictures of the patient – Day 1 and 2 (Dr Ibrahima A Dembélé – Dr Stéphane L Djeugoué)

The paraclinical assessment showed:

Hemogram:

Normochromic normocytic anemia with a hemoglobin level of 8.1 g/dL, the mean corpuscular volume (MCV) of 86.6 fl; a CCMH at 34.3 g/dL. Erythropenia at 2.830.000/mm3

Inflammatory Assessment:

An inflammatory syndrome with a CRP of 177 mg/L. VS at 87mm at the 1st hour and at 121mm at the 2nd hour. Serum protein electrophoresis revealed hypoalbuminemia (21.9g/L), hyper-alpha1globulinemia (3.8g/L) and hypergammaglobulinemia (15.5g/L) in favor of an inflammatory syndrome; but immunofixation was requested but not performed. Complement dosage showed hypocomplementemia C3 (0.43g/L) and normal C4 (1.59g/L)

Infectious Assessment:

Negative HbsAg, anti-Hbc Ab not done, anti-Hbs Ac negative, HIV1-2 and HCV serologies are negative. Negative thick drop. Blood culture and bacteriological examination of vaginal fluid revealed colistin and chloramphenicol-sensitive *Klebsiella pneumoniae.*

Biochemical Assessment:

Serum creatinine at 25 µmol/l with clearance at 192.92 ml/min. Azotaemia at 9.8 mmol/L. Hypocalcemia at 2.05 mmol/L, magnesium level at 0.70 mmol/L, natremia at 134mmol/L, hypokalémia at 2.03mmol/L. Total protein at 57.6g/L, hyperbicarbonatemia at 35.5mmol/L, 24-hour proteinuria at 0.36g/24h; CPK-MM at 325ui/L.

Immunological Assessment:

Anti-nuclear antibodies (ANA-Screen) at 0.30 (negative); anti-SSA antibodies not done; anti-SSB not done, anti-Sm 6.30ui/mL (negative), anti-U_1RNP at 3.00 ui/mL (negative), anti-native DNA at 10.00 ui/mL (negative). Anti-desmoglein antibodies types 1 and 3 were requested, but were not performed.

Anatomo-Cytopathological Assessment: A skin biopsy to look for active superficial dermo-epidermitis was requested but not performed.

The diagnosis was that of an acute attack of his lupus disease with muscular, hematological, renal and cutaneous involvement of the type of toxic epidermal necrolysis of lupus origin (Lyell-like syndrome) triggered by sepsis with genital entry. Pharmacovigilance investigations have been negative. The initial SLEDAI score at Day1 was 9 points.

Initial treatment included early parenteral nutrition with Perikarbiven 1500Kcal slowly intravenously for 3 successive days. Treatment of the skin with Banéocin® ointments (Bacitracin and neomycin) and application of healing cream based on hyaluronic acid were recommended. Skin care was carried out with baths based on Cytéal® antiseptics and Polyvidone iodine, followed by topical application of lotion based on 2% salicylated vaseline on the scabs. Wound care was performed twice daily and the patient was mobilized 3 times a day. Broad-spectrum intravenous antibiotic therapy based on Chloramphenicol 1g every 12 hours. Prevention of thromboembolic disease was achieved by the administration of enoxaparin 4000iu subcutaneously/24 hours and prevention of stress ulcer by lanzoprazole 40 mg + sodium bicarbonate 300mg 1 sachet/24 hours. Paracetamol 1g every 8 hours + nefopam 20mg every 8 hours. Bolus of methylprednisolone 1g in 250cc of saline 0.9% to flow for 1h30 for 3 successive days then Prednisone 1mg/kg (60mg/day), Calcium tablet 1000mg per day, Potassium tablet 600mg per day, Albendazole tablet 400mg for 3 days and Serum salty 0.9% one liter per 24 hours. Daily supportive psychotherapy and therapeutic education for the patient and those around her.

The evolution was marked by an improvement in skin lesions (Image 2) and a regression of facial puffiness and swelling of both feet. But the patient was still feverish and very painful (pain rated at 8/10 according to the numerical scale), she complains of insomnia falling asleep, anxiety. The treatment was to continue with twice-daily wound care, a multimodal analgesic with injectable tramadol 100mg associated with nefopam 40mg in 250cc of 0.9% saline and paracetamol 1g every 6 hours, corticosteroid therapy associated with adjuvant measures continued; amitriptyline 50mg in 200cc of saline solution 0.9% to be infused for 1 hour in the evening before going to bed with and enhanced supportive psychotherapy. Parenteral nutrition was maintained associated with IV rehydration 1500mL per day. The SLEDAI score at Day 13 at 11 points.

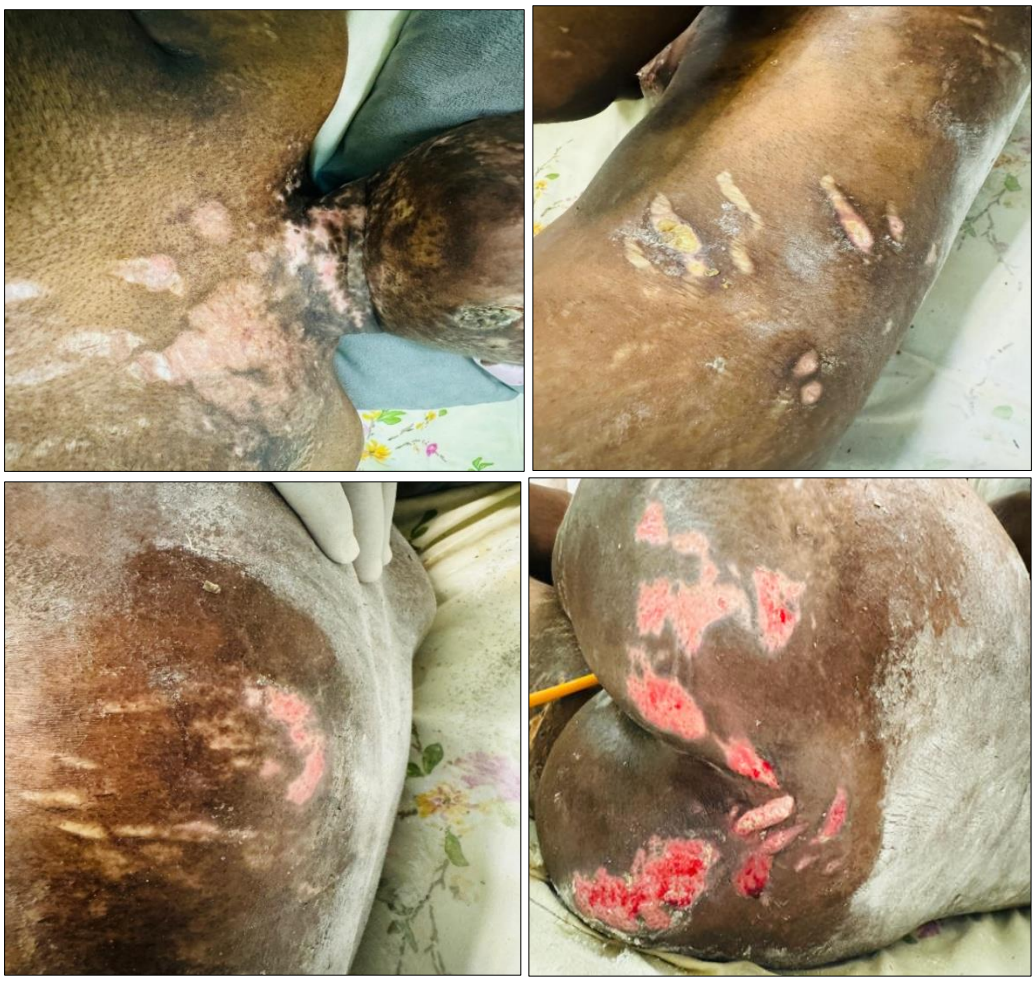

Image 2: Pictures of the patient – Day 17 (Dr Ibrahima A Dembélé – Dr Stéphane L Djeugoué)

After 1 month and 20 days of treatment, the evolution was marked by an improvement of the skin lesions almost all at the re-epithelialization stage, a resumption of oral feeding and an improvement in mobilization; but the patient is still feverish and painful, she presented a melancholic depression associated with a brief psychotic state suggesting psychiatric disorders secondary to lupus disease or corticosteroids for which. She was put on amitriptyline oral drops, 10 drops in the evening and 5 drops in the morning associated with a psychological interview. She continued with corticosteroid therapy, adjuvant measures and the cessation of chloramphenicol; the medical team accentuated therapeutic education with the family.

Image 3: Pictures of the patient – Day 48 (Dr Ibrahima A Dembélé - Dr Stéphane L Djeugoué)

The patient was discharged on Day 50 with treatment of prednisone 60 mg per day associated with adjuvant measures, amitriptyline oral drops, 10 drops in the morning and 5 drops in the evening, strict guidelines on the care of remaining skin lesions, alvityl tonus 1 effervescent tablet in the morning before breakfast, reinforcement of her therapeutic education. The patient recurred skin ulcers predominantly gluteal, submammary and on the scalp; an accentuation of psychiatric disorders following the non-administration of her corticosteroid therapy by her family. She presented with a picture of acute adrenal insufficiency at home and the evolution was fatal.

DISCUSSION

Lupus toxic epidermal necrolysis represents a hyperacute and exceptional cutaneous expression of lupus disease [1-3]. Epidermal necrolysis can reveal or highlight the evolution of lupus, even if toxidermias are more frequent in patients with lupus than in the general population [2]. Since the initial description of the "Lupus-Lyell" syndrome by Mandelcorn in 2003, about a hundred cases have been reported in the literature [4]. Lyell syndrome is a severe acute toxic epidermal necrolysis (TEN) due to an unpredictable idiosyncratic drug reaction, characterized by sudden destruction of the superficial cutaneous-mucosal layer, so the evolution can be fatal [5]. It remains a rare condition with an incidence of 0.1% of the general population [5]. From a pathophysiological point of view, the authors evoke a common concept called "acute pan-epidermolytic apoptosis syndrome" including, in addition to lupus-Lyell and drug-induced Lyell, porphyria cutanea tarda-Lyell and graft-versus-host disease-Lyell [4].

Non-drug epidermal necrolysis accounts for approximately 10% of cases of epidermal necrolysis, and is of infectious, autoimmune (lupus) or idiopathic origin [7]. Epidermal necrolysis can reveal or highlight the development of lupus, even if drug eruption is more common in patients with lupus than in the general population [7]. Compared to Stevens-Johnson syndrome, TEN is extensive over 30% of the body surface, associated with systemic involvement and its higher severity would make its life prognosis serious in its acute phase [5]. In our patient, the extension of epidermal detachments over a surface area of 58.5% argues in favor of Lyell syndrome. The occurrence of generalized bullonecrotic detachments in a systemic lupus setting poses a diagnostic dilemma [4]. The problem is to distinguish between an authentic severe and threatening drug eruption falling within the spectrum of Stevens-Johnson syndrome/Lyell syndrome and a hyperacute expression of lupus disease, especially since lupus subjects are more likely to develop severe bullous drug eruption than the general population [4].

The distinction between drug eruption and a lupus manifestation is therefore difficult: the photodistributed and then secondarily generalized nature of the lesions, an annular appearance, the rarity of mucosal involvement and the progressive evolution over several days with few scarring sequelae point towards the diagnosis of lupus epidermal necrolysis [3]. The negativity of the pharmacovigilance survey, the more or less progressive onset, the initial predominance of lesions in photo-exposed areas, the mild mucosal involvement, the coincidence with a flare-up of lupus disease and the rapid improvement under treatment are the arguments that would argue more in favour of the diagnosis of Lupus-Lyell than of a drug-induced Lyell in our patient. Histology is not very discriminatory, showing interface dermatitis and keratinocyte necrosis, with often negative immunofluorescence [1]. On the biological level, the majority of patients have positive anti-SSA antibodies, and the signs of biological activity of lupus are inconsistent [1, 2], systemic involvement, particularly hematological (cytopenias), is frequently found [1,2].

Multisystem involvement in Lupus-Lyell requires multidisciplinary management in an intensive care unit for major burns with highly qualified support in intensive care and restrictive nursing care, to reduce the mortality rate below 20%. The case that is the subject of our study was managed in the Internal Medicine department of the Point G University Hospital, which does not have an intensive care unit for major burns but is very accustomed to this type of pathology, the initial management was early, rapid and effective and multidisciplinary; which improved the clinical picture of the patient during hospitalization. The management of this disease remains poorly codified, but generally relies on systemic corticosteroid therapy, hydroxychloroquine, immunosuppressants or polyvalent immunoglobulins [1].

The management of Lupus-Lyell, like that of classic systemic lupus, remains a major challenge [5]. This syndrome is characterized by periods of remission; exacerbations and a variable response to treatment even within the same individual [5]. Therapeutic education of the patient and his family must be essential because it is a chronic pathology evolving in flare-ups; as well as the application of hygienic-dietary measures linked to corticosteroid therapy and its non-abrupt discontinuation; the strict salt-free diet, the sugar-free and low-potassium diet [1]. Our patient and her family benefited during her hospitalization and during the various health checks from therapeutic education on the clinical manifestations of systemic lupus, its complications, the objective of the therapy; as well as daily supportive psychotherapy associated with major anxiolytics. Despite this, the family did not follow the instructions concerning corticosteroid therapy and it was stopped several times at home. This was aggravated by the psychiatric disorders that the patient presented with opposition to any therapy, leading to a re-accentuation of the skin lesions, acute adrenal insufficiency and the fatal evolution of the patient.

The large skin detachment constitutes a huge gateway and the prevention of infection is based on the application of very strict aseptic measures such as the isolation of the patient in order to reduce the risk of cross-contamination and strict asepsis of the nursing staff. Thus, in our patient all care procedures were administered in compliance with the rules of aseptic technique involving the wearing of masks, sterile gloves, caps and bibs without the notion of isolation, but she was hospitalized in a private room with a limitation of the number of visitors. Since the patient was not isolated, the increased risk of cross-contamination justifies the massive administration of broad-spectrum antibiotics from the first days of hospitalization by direct intravenous (IVD).

Nutritional intake is of great importance in the treatment of Lupus-Lyell [5]. Artificial nutrition currently aims to limit visceral damage and reduce morbidity and mortality by modulating the inflammatory and immune response, and by limiting stress [5,]. Consensus or expert conferences have not specified the optimal time to initiate enteral nutrition (EN) [5]. It is immediate when initiated within the first six hours of hospitalization, early, when introduced between 24 and 48 hours, and conventional, after the 3rd day [5]. EN has several advantages over parenteral nutrition, it preserves trophicity and digestive motility, reduces bacterial and toxin translocation, and reduces secondary enteral complications.

In cutaneous lupus, local glucocorticoids used are desonide 0.5% on the face once daily for a short period (risk of atrophy on the face), betamethasone dipropionate or clobetasol for the (scalp and integument off the face) [1-13]. Systemic glucocorticoids are often used for extracutaneous involvement of LS and seem to be more effective for acute and subacute cutaneous involvement than discoid. For cutaneous involvement, general corticosteroid therapy should be given for the shortest possible period by weighing the benefit/risk but can sometimes be useful to control inflammatory lupus lesions [1-13]. The use of corticosteroids seems particularly interesting for lupus panniculitis at the start of treatment [1]. In our observation, the patient received systemic corticosteroid therapy from the outset in bolus then in maintenance by oral route, associated with adjuvant measures, which significantly improved her general condition and the skin lesions.

The choice of systemic treatments depends on the severity of the skin lesions which can be assessed by the CLASI activity score (Cutaneous Lupus Erythematosus Disease Area and Severity Index): low CLASI activity 0 - 9; moderate: 10 - 20, severe > 20, the patient's request (shared medical decision); the risk of scarring, other systemic lesions, the desire for pregnancy and other comorbidities [1]. Hydroxychloroquine (HCQ) and chloroquine (CQ) are used in case of failure of local treatments or immediately, in combination in case of moderate to severe forms: At doses of 6.5 mg/kg/day for HCQ and 3-4 mg/kg/day for CQ, a clear clinical improvement in more than 60% of cases. Full efficacy can be judged after 3 to 4 months of treatment [1-13]. Our patient had not been initially put on HCQ because she had a history of maculopathy.

In case of contraindications to antimalarials, methotrexate, Thalidomide, Anifrolumab or Belimumab are used as 2nd line [13]; in case of resistant skin lesions, Mycophenolate mofetil is an alternative [13]. Azathioprine is indicated for severe forms of LS, in patients who are intolerant to corticosteroids or corticosteroid-dependent or whose therapeutic response is insufficient despite high doses of corticosteroids [13]. Lenalidomide in cases of cutaneous lupus refractory to first- and second-line treatments or in cases of intolerance to thalidomide in combination with aspirin at anti-platelet doses [13].

CONCLUSION

The diversity of skin lesions in systemic lupus should suggest the diagnosis of lupus toxic epidermal necrolysis in the presence of skin lesions suggestive of toxidermia without the notion of suspicious drug intake. Lupus toxic epidermal necrolysis is an exceptional entity, the exact mechanism of which is not yet well understood, it represents a significant diagnostic challenge due to its severity, the therapeutic urgency and the impact on management. Stevens Johnson/Lyell syndrome constitutes the real differential diagnosis due to the clinical similarity at all points and must be made quickly to improve the prognosis.

Consent: Written informed consent was obtained from the patient's family to publish this report in accordance with patient consent policies.

Competing Interests: The authors declare no conflict of interest.

Authors' Contributions: All authors participated in the evaluation and follow-up of the patient, in the writing and correction of the case report. All the authors of the manuscript have read and agreed to its content

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