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CASE REPORT

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Staphylococcal scalded skin syndrome in adults with obesity and type 2 diabetes: A case series

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Abstract

Staphylococcal scalded skin syndrome (SSSS) primarily affects children and rarely adults with immunodepression. We describe two cases of adults diagnosed with SSSS with no additional cause of immunological compromise other than obesity and uncontrolled diabetes. An increased risk of infection should be considered in cases of obesity and diabetes.

KEYWORDS

case report, immune system dysregulation, immunodepression, obesity, staphylococcal scalded skin syndrome, type 2 diabetes

1 | INTRODUCTION

Staphylococcal scalded skin syndrome (SSSS) is a skin disorder produced by a bacterial toxin that typically affects children and immunocompromised adults. The cutaneous lesions appear as flaccid bullae that progress to scaly and erythematous skin.[1]

We present two cases of adults with no known immunological deficit but obesity and type 2 diabetes, highlighting the need of not minimizing the risk of infection in a metabolically dysregulated patient.

2 | CASE REPORTS

2.1 | Patient 1

A 58-year-old woman was admitted to our hospital's emergency department (ED) due to the development of a widespread, itchy erythema and general malaise. The patient mentioned an all-day solar exposure with no skin protection for UV rays 2 days before the onset of symptoms and a shellfish-based dinner the evening before. Also, she reported paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs) abuse (20–25 tablets of various drugs in only 7 days, specific doses not recalled) during the week before due to non-specific pain in right foot, not attributable to history of trauma. She described that the scaly erythema developed progressively in the 48 hours preceding the ED admission. At first, it presented as a macular erythema with skin soreness; then, it became more prominent in skin folds and flaccid bullae appeared.

Patient's past medical history included type 2 diabetes (T2D), diagnosed 10 years earlier and in treatment with a glucagon-like peptide 1 receptor agonist (GLP1-RA) and long-acting insulin, II class obesity (body mass index, BMI 35 kg/m^2), arterial hypertension, dyslipidemia, hyperuricemia, carotid artery disease (with 30% and 40% stenosis

of right and left carotid arteries, respectively), previous ischemic transient attack, and herpes simplex-virus related encephalitis 5 years earlier. Her medical records were also noteworthy for Charcot foot. Her medications included pantoprazole, allopurinol, salicylic acid, bisoprolol, simvastatin, furosemide, perindopril/amlodipine, and the association degludec insulin/liraglutide.

On arrival at the ED, the patient presented a disseminated erythematous rash covering most of the body surface with diffuse desquamated areas particularly evident at the level of neck, armpits, and groin. The lesions initially presented as flaccid bullae with initial desquamation leaving patches of erythematous skin. Moreover, the patient presented radial fissuring around the nose and the mouth. Blood pressure (BP) was 140/70 mmHg, axillary temperature was 36°C, heart rate 92 beats per minute and oxygen saturation 98% in ambient air. On blood testing, a deterioration in the baseline renal function was noted, with increased levels of serum creatinine (1.6 mg/dl) and blood urea nitrogen (210 mg/dl), suggesting a prerenal cause of acute kidney injury. Blood glucose was 311 mg/dl. There was a rise in transaminases (AST 351 U/L, ALT 433 U/I), alkaline phosphatase (ALP, 1463 U/I), bilirubin (total 1.32 mg/dl, direct bilirubin 0.99 mg/dl), gamma-glutamyl transferase (γGT) 1628 U/I, and uric acid (8.8 mg/dl). There was an alteration of the electrolytes with hyponatremia (Na^+ 127 mEq/L) and mild hyperkalemia (K^+ 4.9 mEq/L). C-reactive protein (CRP) was only slightly increased (16 mg/L) and complete blood count showed only a rise in the white blood cell (WBC) count (10,720 per mcl) of which 54.5% were represented by neutrophils and 20.7% by lymphocytes.

Because of the hypertransaminasemia associated with cholestasis, paracetamol was discontinued, and an abdominal ultrasound (US) was performed, revealing normal liver dimension and mild diffuse increase in liver echogenicity compared with the kidney, consistent with fatty liver disease. It also revealed the presence of biliary sludge in the gallbladder.

After 2 days in ED, the patient was admitted to our Internal Medicine Unit for additional diagnostic testing and treatment. At admission, BP was 150/80 mmHg, axillary temperature was 36.1°C, heart rate 80 bpm and the oxygen saturation 98% in ambient air. The patient height was 165 cm and body weight 95.6 kg (BMI 35 kg/m^2). The patient presented a scaly erythema diffused to the whole-body surface, particularly intense at the level of the skin folds. There were crusting and fissuring near the mouth, the nose, and the eyes. The blistering and the successive desquamation leaved regions of dump and shiny skin, while mucous membranes were unaffected. Breath sounds were reduced at the bases of the lungs. Heart sounds were normal. Bilateral peripheral edema was present. A structural deformity and a periungual wound of the right foot were noticed and were attributed to Charcot osteoarthrosis. Abdominal examination was normal. Blood tests revealed progressive resolution of paracetamol-induced hepatitis (AST 35 U/I, ALT 121 U/I, bilirubin 0.61 mg/dl, and ALP 663 U/I), as well as a complete resolution of prerenal acute kidney injury (serum creatinine reduced to 0.86 mg/dl and blood urea nitrogen to 48 mg/dl). Severe metabolic alterations were observed, which included dyslipidemia (total cholesterol 224 mg/dl, HDL-cholesterol 27 mg/dl, and LDL-cholesterol 133 mg/dl), hypertriglyceridemia (432 mg/dl), and hyperglycemia (Hb1Ac 81 mmol/mol). CRP remained only slightly increased (8.9 mg/L). Urinalysis revealed increased leukocytes (500 per mcl), esterase (500 per mcl), absence of nitrites, and presence of glucose (100 mg/dl). Anti-nuclear antibodies (ANA) and extractable nuclear antigens (ENA) tests were negative, and no other evidence of autoimmune disease was found. To further investigate the foot deformity, magnetic resonance imaging (MRI) was performed, which revealed signs of osteonecrosis of tarsal bone elements and plantar fasciitis.

In the initial suspicion of a serious cutaneous allergic reaction to NSAIDs, a steroid-based treatment was initiated with intravenous methylprednisolone 40 mg once daily, but the lack of improvements in clinical presentation and the concerns about using steroids in a decompensated diabetic patient prompted us to discontinue the therapy.

On the third day after admission, the patient presented high fever (39°C), cough, and vomiting. Vital signs showed high-rate tachycardia (150 bpm), rapid respiratory rate (25 bpm), hypotension (BP 95/50 mmHg), and low peripheral oxygenation (SpO2 74% in ambient air). Considering the history of long-duration and poorly controlled T2D, to evaluate the possibility of myocardial infarction with atypical signs, an electrocardiogram (EKG) was performed, and it showed sinus tachycardia and a change in repolarization with ST-depression in the anterior-inferior leads. Cardiac biomarkers were tested, and troponin I progressively increased, from 32.5 ng/L to 1759 ng/L and to 5973 ng/L, respectively, at 3 and 6 h. A chest X-ray was performed, and it revealed thickening of interlobar fissures and upper lobe pulmonary venous diversion, findings suggestive for pulmonary edema. Acute decompensated heart failure was treated with intravenous diuretics and high-flow oxygen therapy. Meanwhile, blood examinations showed increased inflammatory indices, with CRP peaking to 122 mg/L. Blood cultures from peripheral vein were collected and sent to the laboratory, and an empiric antibiotic regimen with piperacillin/tazobactam 4.5 g intravenous three times a day was started. The next day, an echocardiography was performed, which revealed low left

ventricular ejection fraction (EF 40%) and a septal and right ventricular apex akinesia. Following the isolation of methicillin-susceptible *Staphylococcus aureus* (MSSA) in blood cultures, the antibiotic therapy was shifted to oxacillin 3 g intravenous four times a day, which was continued for 14 days. The rash gradually faded in a few days. A coronary angiography was performed, with angioplasty and stenting of the anterior descending coronary artery and of the right coronary artery. A transesophageal echocardiography and a nasal swab were performed to find the source of the sepsis, but both resulted negative. The infection's source was then attributed to the lesion in the right foot.

A week after starting antibiotics, the skin sores had completely healed, the blood cultures were found negative, and the patient showed no signs or symptoms of infection. In terms of cardiac function, the patient recovered entirely. Indeed, transesophageal echocardiography demonstrated that the left ventricle's contractility had been recovered and that there were no thrombotic lesions.

The final diagnosis was SSSS, which occurred in the context of an increased susceptibility to infections due to the patient's history of obesity and poorly controlled T2D.

2.2 | Patient 2

A 54-year-old man presented to our hospital's emergency department complaining acute back pain that had been interfering with his daily activities for 15 days, not responding to NSAIDs, fever (39.7°C) and scaly erythematous lesions on both elbows with early desquamation, on legs and on feet. Patient's past medical history included class 2 obesity (BMI of 35.9 kg/m^2) and untreated arterial hypertension.

At admission, elevation of acute phase proteins (CRP 343 mg/L and procalcitonin 3.47 mg/dL) and of WBC count (14,000 per mcl) on blood testing suggested the presence of an infection. A lung CT scan was performed to investigate the infection's source, and pneumonia was ruled out. In addition, because of the acute back pain, a vertebral bones CT scan was performed, with radiologic evidence of spondylotic arthrosis and Schmorl's nodes in D11 and L1. In the clinical suspicion of cellulitis, an empiric antibiotic therapy with daptomycin was initiated. After 72 h, the skin sores continued progressing in gravity and extension, while creatinphosphokinase (CPK) levels increased. Due to lack of efficacy and muscle toxicity, daptomycin was discontinued and vancomycin was introduced.

After 4 days in the emergency department, the patient was admitted in our Internal Medicine Unit. At the time of admission, patient's blood pressure in clinostatism was 140/80 mmHg, his axillary temperature was

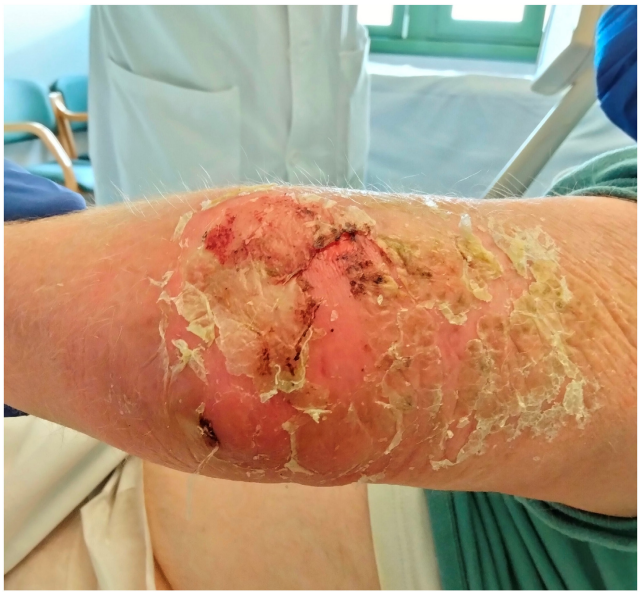

FIGURE 1 Erythematosus and desquamative lesions of left elbow of Patient 2, at admission.

36.6°C, his heart rate was 90 beats per minute, and his oxygen saturation was 93% while breathing ambient air. BMI was 36 kg/m^2. The patient presented erythematosus and desquamative lesions on both elbows (Figure 1) and ankles. He also suffered of radial fissuring on perioral skin, without mucous membrane involvement. Breath sounds were diminished at the lungs' bases. He was tachycardic, and a systolic 2/6 Levine heart murmur was noticed. Both legs presented bilateral peripheral edema. Abdominal examination was normal. Blood tests showed hemoglobin at the lower limits of normal values (12 g/dl) and WBC were 11,600 per mcl (of which neutrophils 87.4% and lymphocytes 9.3%). Blood tests revealed no alteration in electrolyte balance, a slight increase in liver enzymes (ALT 68 U/L and AST 85 U/L), significant systemic inflammation (CRP 126 mg/L, ferritin 4007 ng/ml, and D-dimer 4476 ng/ml), diabetes mellitus (Hb1Ac 49 mmoL/L) and findings suggestive for chronic malnutrition (plasmatic iron 19 mcg/dl, total cholesterol 77 mg/dl, and LDL-cholesterol 41 mg/dl). Patient tested negative for HIV, and serum protein electrophoresis excluded hypogammaglobulinemia. Blood cultures were collected from peripheral vein during fever, and MSSA was identified. Antibiotic therapy was shifted to oxacillin according to antibiogram. Because the low back pain was not responding to analgesic therapy, in the suspect of spondylodiscitis, a vertebral column MRI was performed, which confirmed the CT evidence of some discal protrusions, a Schmorl's node of D11, and a collapsed L2. MRI revealed no signs of spondylodiscitis.

During hospitalization, the patient developed subcutaneous and intramuscular abscesses in the right elbow,

right hip flexor, and left abductor magnus. The two subcutaneous abscesses spontaneously fistulized to the skin surface and the purulent secretions were sent for microbiological analysis which identified MSSA. Also, left abductor magnus abscess was surgically drained and MSSA again isolated. In the clinical suspicion of endocarditis, a positron emission tomography–computed tomography (PET-CT) and a transesophageal echocardiography were conducted to identify possible valvular vegetation or paravalvular abscesses, but both tests revealed no evidence of valvular involvement. The pattern of radiopharmaceutical accumulation demonstrated abnormal uptake in subcutaneous and muscular tissue of upper and lower limbs, with higher uptake in the locations where abscesses developed. The cutaneous involvement gradually healed after 2 weeks of antibiotic treatment with oxacillin 3 g intravenous four times a day. On the contrary, abscesses showed no evidence of improvement, so antibiotic therapy was changed in accordance with the infectious disease specialist in favor to linezolid 600 mg intravenous twice a day, then, after 2 weeks, to a single dose of dalbavancin 1500 mg intravenous, with satisfying response.

The final diagnosis was SSSS and pyomyositis in a patient with obesity and new-onset diabetes determining an immune-compromised status.

3 | DISCUSSION

Staphylococcal scalded skin syndrome, also known as Ritter disease, is a disease characterized by denudation of the skin caused by the epidermolytic (or exfoliative) exotoxin produced by some strains of *Staphylococcus aureus*, typically from a distant site. SSSS is characterized by rapidly spreading, extensive cutaneous erythema made of flaccid, sterile bullae, erosions, and sheet-like desquamation without mucosal involvement.[1] Skin pain, fever, and irritation are also present, as well as thick crusts and radial fissuring around mouth, nose, and eyes.

Skin lesions are generated by the hematologic dissemination of exotoxins from the site of infection to the skin. In both cases we presented we searched for the source of the infection. In the first case, we identified it in the right foot wound, while in the second case, we were not able to determine which lesion came first.

The management of SSSS implies the eradication of the infection and supportive care, so that it usually requires hospitalization. Intravenous treatment with an antistaphylococcal antibiotic, usually a penicillinase-resistant penicillin (for instance oxacillin or nafcillin) or a 2nd or 3rd generation cephalosporine or vancomycin should be started promptly. The total duration of therapy is typically 10–14 days. Mortality is less than 10% in children, but it rises to 40–60% in adults, despite antibacterial therapy.[2] Death is usually determined by complications such as electrolyte imbalance or sepsis.

Staphylococcal scalded skin syndrome is less common in adults than in children, because most of adults have antibodies to the Staphylococcal exotoxin. Moreover, adults are more likely to be affected in case of concomitant serious illness or immunodeficiency. Immunodepression (including acquired or genetic immunodeficiency syndrome, long-term systemic corticosteroid therapy, or systemic therapy with cytostatic or immunosuppressive drugs), cancer (including non-Hodgkin's or Hodgkin's disease, leukemia, or solid neoplasms), and kidney failure are all known risk factors for SSSS in adults.[2–4]

In both patients, the symptom that guided the diagnosis was the desquamated, scaly erythematous skin, without mucosal involvement. When the Patient 1 arrived at the ED, she mentioned three possible (and likely) causes: sunburn, an allergic reaction to shellfish, drugs assumption side effects. The differential diagnosis was made both on the clinical presentation and on the time of onset of symptoms. Firstly, the sunburn and the allergic hypotheses were ruled out. In fact, the sun exposure did not result prolonged enough to induce such lesions, but, mostly, the skin sores involved also non-exposed body areas like palms, plants, armpits, and inguinal folds. Similarly, the timing of symptoms onset allowed to rule out an IgE-mediated response. Skin reactions are rarely related to paracetamol use, and previous use of this drug was not associated with any cutaneous manifestation. NSAIDs, on the contrary, are very infrequent cause of type 3 or type 4 allergic reactions, which often present as urticaria, vasculitis, Steven Johnson syndrome (SJS), or Lyell syndrome (also known as toxic epidermal necrolysis, TEN). On the basis of the features of the skin lesions—desquamated, scaly, strongly erythematous skin without mucosal involvement—both SJS and TEN could be excluded, as well as bullous pemphigoid.

Steven Johnson syndrome and TEN are severe blistering eruptions of the skin and mucous membranes usually caused by medications. A cutaneous biopsy should be performed to support the diagnosis to demonstrate subepidermal blistering and necrosis. SJS and TEN are invariably associated with mucosae, which are unaffected in the case of SSSS.[5]

Pemphigus foliaceus and pemphigus vulgaris are blistering autoimmune diseases. Pemphigus foliaceus, like SSSS, is characterized by superficial blistering and a lack of mucosal involvement, but systemic symptoms are generally absent, whereas our patients had significant malaise and fever. In pemphigus vulgaris, blistering occurs lower in the epidermis, resulting in deeper erosions and

distinctive mucosal erosions, which, as previously mentioned, were absent in our patients.[6]

Streptococcal and staphylococcal toxic shock syndromes, which are toxin-mediated systemic bacterial infections that might mimic SSSS, were also ruled out as a differential diagnosis. In most cases, the causing infection is an abscess, or superinfection with a retained foreign body. In contrast to SSSS, there is no perioral crusting, no bullae or desquamation, and a negative Nikolsky sign.[7]

Another possible condition that could cause skin lesions was the cutaneous T-cell lymphoma, but this was considered incompatible with these specific dermatological traits by a consultant dermatologist. Indeed, although itch is a frequent symptom that affects up to 80% of patients, cutaneous T-cell lymphomas typically present as patches, papules, nodules, plaques, and/or evident tumors.

However, only when Patient 1 developed fever and laboratory values were consistent with infection blood cultures were collected and *Staphylococcus aureus* was finally identified as the responsible of SSSS. In fact, for Patient 2, an intercurrent infection was evident since the admission in ED and this led to a faster identification of MSSA, even if the symptom for which the patient presented to ED (low back pain) was apparently not related to the infection.

Both our patients did not have personal or familiar history of known immunodeficiency, neither immunosuppressive treatment nor prolonged serious illness that could affect immune response. As previously mentioned, both patients suffered from T2D and obesity. The susceptibility to infection in diabetes is well known, especially in a context of poor glycemic control.[8] Previous studies have shown that diabetes increased susceptibility to infections is related to neutrophil dysfunction (including impaired chemotaxis, phagocytic abilities, and lower microbicidal activities) or to disturbances in T-lymphocyte responses to infection.[9–12] Other possible causal pathways result from common complications of diabetes, such as neuropathy and vascular insufficiency.[13] T2D has been described as closely related to an increased risk of community-acquired *Staphylococcus aureus* bacteremia,[14] but very few examples of SSSS in adults caused by *Staphylococcus aureus* are reported.[15,16]

Several epidemiological data suggest that also obesity is associated with an increased risk of infection.[9,17] Adipose tissue can be considered an endocrine organ in all respects as it produces adipokines with local and systemic effects.[18] In patients with obesity, the dysfunction of adipose tissue determines an imbalance favoring the increase of pro-inflammatory adipo-cytokines (such as leptin, resistin, interleukin-6, tumor necrosis factor-α, and others).[19] This determines a chronic low-grade inflammation, interfering with immune response (both innate and adaptive immunity) and making obese patients more susceptible to develop infections.[20,21]

4 | CONCLUSION

Staphylococcal scalded skin syndrome should be considered when evaluating a scaly non-mucosal erythema in adults with no obvious immunodeficiency etiology but with a subtle, albeit substantial, risk of infection due to diabetes and obesity.

Diabetes-related vulnerability to infection is widely established clinically and evidence regarding immunological dysregulation in obesity is growing,[22] despite mechanisms are still under discussion. This case series emphasizes the need to consider a metabolically unhealthy patient such as a non-totally immune-competent patient.

AUTHOR CONTRIBUTIONS

Carolina Vitale and Valentina Spinozzi was the patient's clinician, reviewed the literature, and drafted the manuscript. Colangeli Luca contributed to manuscript drafting and revision. Paolo Sbraccia was responsible for the revision of the manuscript. Valeria Guglielmi was the patient's clinician and contributed to manuscript drafting. All authors issued final approval for the version to be submitted.

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CONFLICT OF INTEREST

The authors declare that they have no competing interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

CONSENT

We confirm that a written informed consent from patients was obtained before the submission to the journal.

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