ATOPIC DERMATITIS IN AN INFANT: PRESENTATION OF A CLINICAL CASE WITH SECONDARY STAPHYLOCOCCUS AUREUS INFECTION

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Abstract

Atopic dermatitis (AD) is a chronic inflammatory skin disease with a high prevalence in the pediatric age group, characterized by xerosis, intense pruritus, eczematous lesions, and a predisposition to secondary bacterial infections, mainly with Staphylococcus aureus. Its pathogenesis is complex and involves dysfunction of the epidermal barrier, immunological disturbances polarized toward a Th2 response, and changes in the skin microbiome. This paper presents the case of a 6-month-old male infant who was referred to the Department of Dermatology of Tetovo Clinical Hospital with erythematous eczematous lesions on the cheeks, marked pruritus, subfebrile temperature, and clinical signs of secondary bacterial infection. The infant had a strongly positive family history of atopy: his grandmother had atopic dermatitis, his maternal uncle had allergic rhinitis and bronchial asthma, while his mother had episodes of contact dermatitis. The diagnosis was established on the basis of clinical examination, dermatoscopic assessment, and a bacterial culture obtained by swab from the lesions, which confirmed the presence of methicillin-sensitive Staphylococcus aureus (MSSA). The therapeutic plan included detailed parental education on skin care, frequent use of a hypoallergenic emollient containing ceramides, hydrocortisone cream 1% twice daily for 10–14 days on inflammatory lesions, and mupirocin 2% ointment three times daily for 7 days for the bacterial infection. At the follow-up visit after two weeks, considerable clinical improvement was noted, with regression of erythema, elimination of the honey-yellow crusts, and reduction of pruritus. This case underscores the importance of early diagnosis, an individualized therapeutic approach, and caregiver education for the effective management of AD in infancy, especially when complicated by secondary bacterial infections.

Keywords: atopic dermatitis, infancy, Staphylococcus aureus, epidermal barrier, ceramides, hydrocortisone, mupirocin.

Introduction

Atopic dermatitis (AD), also known as atopic eczema, is one of the most common chronic inflammatory skin diseases in the pediatric population. According to contemporary epidemiological estimates, this disease affects between 10% and 20% of children worldwide, with an increasing trend in recent decades, especially in industrialized countries and urban centers (Bylund et al., 2020; Silverberg, 2017). Studies in the Balkan region and in Southeastern Europe show similar trends, making AD a significant public health problem and one of the most frequent reasons for pediatric dermatology visits (Weidinger et al., 2018).
The clinical manifestations of AD usually begin early in life: about 60% of patients develop symptoms within the first year of life, while up to 90% do so before the age of 5 (Weidinger et al., 2018). The clinical picture is heterogeneous and varies according to age, severity, and localization. In infants, the lesions mainly involve the cheeks, the scalp, the extensor surfaces of the extremities, and the trunk, while areas covered by the diaper are usually spared because of the increased local hydration of this area (Eichenfield et al., 2014a). The characteristic lesions include erythematous macules, papules, vesicles, exudation, crusts, and marked xerosis. Pruritus, the cardinal symptom of the disease, deeply affects the quality of life of the child and

the family, causing sleep disturbances, mood changes, and a notable psychosocial impact (Silverberg & Simpson, 2014).

The pathogenesis of AD is a multifactorial process resulting from the close interaction between genetic, immunological, environmental, and microbial factors. Three main pathogenetic axes are currently recognized: dysfunction of the epidermal barrier, immunological dysregulation, and disturbances of the skin microbiome (Weidinger et al., 2018; Boguniewicz & Leung, 2011).

Filaggrin, an essential structural protein of the stratum corneum of the epidermis, plays a key role in maintaining the integrity of the cutaneous barrier and in skin hydration through the formation of natural moisturizing factors (NMF). Loss-of-function mutations in the *FLG* gene are considered the most important genetic predisposing factor identified to date for atopic dermatitis (AD), being reported in approximately 20–50% of patients with moderate to severe forms of the disease (Palmer et al., 2006; Irvine et al., 2011). The deficiency of filaggrin and its products leads to marked transepidermal water loss (TEWL), to an increase in the surface pH of the skin, and to increased permeability of allergens and pathogens through the damaged horny layer (Irvine, 2011).

From an immunological standpoint, atopic dermatitis (AD) is characterized by an immune response polarized toward type 2 helper T lymphocytes (Th2), which produce elevated levels of proinflammatory cytokines such as interleukin-4 (IL-4), IL-5, IL-13, and IL-31. These cytokines contribute to chronic inflammation of the skin, damage to the cutaneous barrier, and the appearance of the pruritus characteristic of the disease (Brunner et al., 2017; Weidinger & Novak, 2016). IL-4 and IL-13 promote IgE synthesis, inhibit keratinocyte differentiation, and suppress the expression of endogenous antimicrobial peptides such as beta-defensin and cathelicidin, while IL-31 is one of the principal mediators of pruritus (Czarnowicki et al., 2019). In the chronic stages of the disease, there is also a significant contribution from the activation of the Th22 and Th17 axes, which explains the evolution from acute exudative lesions toward thickened, lichenified plaques (Boguniewicz & Leung, 2011; Leung & Guttman-Yassky, 2014).

Normal skin is populated by a rich diversity of commensal microbes that exert a protective effect against colonization by pathogens. In patients with AD, this microbial balance is disrupted. Numerous studies have shown that the skin of patients with AD is colonized by Staphylococcus aureus in about 70% of cases with active lesions and up to 90% during flares, compared with only 5–30% on the skin of healthy individuals (Geoghegan et al., 2018). S. aureus is not merely a transient colonizer but plays an active pathogenic role through the production of superantigens, toxins, and proteolytic enzymes that amplify inflammation and further damage the barrier (Mempel et al., 1998; Geoghegan et al., 2018).

In addition to these intrinsic factors, environmental factors such as urbanization, air pollution, limited exposure to microbial diversity in early childhood (the hygiene and biodiversity hypotheses), dietary changes, and the excessive use of soaps and aggressive detergents play an important role in modulating the onset and severity of the disease (Flohr & Mann, 2014).

Atopic dermatitis (AD) is often accompanied by other atopic diseases, a phenomenon known as the "atopic march," which describes the progression of allergic manifestations from atopic dermatitis in early childhood toward the development of bronchial asthma and allergic rhinitis later in life. This phenomenon reflects the complex interaction between genetic predisposition, damage to the cutaneous barrier, and immunological dysregulation (Spergel & Paller, 2003; Zheng et al., 2011). Early identification and adequate treatment of AD may contribute to

modifying this natural course. The management of AD requires a comprehensive approach that includes: education of the patient and the family, restoration of the epidermal barrier with emollients, control of inflammation with topical corticosteroids or calcineurin inhibitors, identification and avoidance of trigger factors, and adequate management of secondary infections (Eichenfield et al., 2014b; Wollenberg et al., 2018).

Aim of the Paper

The aim of this paper is to present and analyze a clinical case of atopic dermatitis with secondary bacterial infection in a 6-month-old infant, and to discuss it in the context of the contemporary literature. More specifically, the objectives of the paper are:

- to describe the clinical, anamnestic, and demographic features of a characteristic case of AD in infancy;
- to illustrate the diagnostic process and the role of complementary examinations, including dermatoscopic assessment and bacterial culture;
- to underscore the importance of parental education and of an integrated approach in the long-term management of AD;
- to compare the clinical findings and therapeutic results of the case with the data published in the current scientific literature.

3. Presentation Of The Clinical Case

3.1. Anamnestic and demographic data: The patient is a 6-month-old male infant who presented at the Department of Dermatology of Tetovo Clinical Hospital, accompanied by his parents, because of marked lesions on the skin of the face, accompanied by intense pruritus and clinical signs suggestive of a secondary bacterial infection. According to the parents' account, the dermatological symptoms had begun approximately two weeks before the visit, with significant worsening over the last 5–7 days.

In addition to the dermatological manifestations, the infant presented with a subfebrile body temperature of 38.2°C and increased signs of discomfort, restlessness, and sleep disturbance. The parents denied the presence of recent viral infections or contact with sick persons in the family circle. They also denied the use of new skin care products and exposure to potential irritants such as aggressive detergents or scented soaps.

Personal history: the infant was born at term through a standard vaginal delivery, with a normal birth weight of 3.4 kg. From birth he had been exclusively breastfed and had developed adequately according to the normal milestones of growth and psychomotor development. At 4 months of age he had experienced a mild episode of an upper respiratory tract infection, which had resolved spontaneously. No other serious infections or previous hospital admissions were reported. Immunizations were up to date according to the national immunization schedule.

The family history was strongly positive for atopic diseases: the maternal grandmother had a documented history of atopic dermatitis, while the maternal uncle suffered from allergic rhinitis and bronchial asthma. The paternal family history was unremarkable for atopic diseases. The mother reported rare episodes of contact dermatitis, but no other significant systemic diseases.

3.2. Physical examination: On dermatological examination, well-defined erythematous plaques were noted on both cheeks, accompanied by mild edema, honey-yellow crusts

(suggestive of staphylococcal infection), and small erosive areas with serous exudation. The extensor surfaces of the arms also showed mild eczematous lesions with visible xerosis. Pruritus was evident from the frequent movements of the infant's hands toward the face and from the marks of self-induced scratching. The mucous membranes were unaffected, and no significant peripheral lymphadenopathy was found. Pulmonary and cardiac auscultation were normal; the abdomen was soft, without organomegaly. Neurological and motor development was assessed as adequate for age.

3.3. Additional examinations and diagnosis: Dermatoscopic examination confirmed the presence of erythematous areas with serous exudation, crusts, and signs of local microinfection. In order to identify the etiologic agent of the secondary infection, a sample was taken by swab from the crusted lesions and sent to the microbiology laboratory. Bacterial culture confirmed the growth of methicillin-sensitive Staphylococcus aureus (MSSA). The antibiogram showed good sensitivity to mupirocin, oxacillin, clindamycin, and trimethoprim-sulfamethoxazole, and no clinically important resistance.

On the basis of the clinical features (typical localization on the cheeks in infants, xerosis, intense pruritus, eczematous lesions), the strongly positive family history of atopy, and the microbiological confirmation of infection with S. aureus, the diagnosis of moderate-to-severe atopic dermatitis, complicated by secondary bacterial infection with Staphylococcus aureus (MSSA), was established. The diagnosis met the modified Hanifin and Rajka criteria for atopic dermatitis (Hanifin & Rajka, 1980).

3.4. Therapeutic plan: The treatment offered was multimodal and individualized according to the patient's age and disease severity, following the principles of current international guidelines:

Parental education. The parents were given detailed instructions on skin care. They were advised to use lukewarm (not hot) water for baths, of short duration (5–10 minutes), with mild detergents, free of fragrances and without classic soap. After bathing, the skin was to be dried by gently patting with a soft towel.

A hypoallergenic, fragrance-free emollient cream containing ceramides and other humectants was prescribed. Application several times daily was advised, especially within 3 minutes after bathing — the technique known as "soak and seal" — in order to maintain maximal skin hydration. This measure addresses the fundamental dysfunction of the epidermal barrier, which is the cornerstone of AD (Sidbury et al., 2014).

For the acute inflammatory lesions, hydrocortisone cream 1% was prescribed, twice daily, for a period of 10–14 days. The choice of hydrocortisone, a low-potency corticosteroid (class VII according to the American classification), was deliberate, given the infant's age and the localization of the lesions on the face — an area with increased skin permeability. This choice provides sufficient anti-inflammatory effect while minimizing the risk of systemic absorption and of local side effects such as atrophy or telangiectasias (Eichenfield et al., 2014b).

After confirmation of the S. aureus infection and the antibiogram results, mupirocin 2% ointment was prescribed for application three times daily for 7 days on the affected and infected areas. Mupirocin shows pronounced activity against gram-positive bacteria, including MSSA and MRSA, and is suitable for use in infants (Bath-Hextall et al., 2010).

A first follow-up visit was scheduled after 7 days, and a second after 14 days, in order to assess the therapeutic response and to identify any unfavorable course at an early stage.

3.5. Clinical course: At the first follow-up, after 7 days of treatment, considerable improvement was noted: the temperature had normalized, the honey-yellow crusts had been substantially reduced, the erythema of the cheeks was paler, and pruritus had decreased according to the parents' subjective assessment. Treatment with mupirocin was discontinued after 7 days, while hydrocortisone was continued for another 7 days on a reduced regimen (once daily). Emollients were recommended for long-term use.
At the second follow-up, 14 days after the start of treatment, almost complete regression of the eczematous lesions was noted. The infant appeared clinically calm, with normalization of sleep and no new signs of cutaneous infection. The parents were advised to continue the regular use of emollients as part of the basic long-term skin care. They were also offered a long-term management plan based on the "step-up/step-down" therapeutic approach, which included the identification and avoidance of potential factors that could trigger worsening of the disease.

Discussion

The case presented illustrates the typical clinical picture of atopic dermatitis in infancy and underscores several important points that deserve detailed analysis in the context of the contemporary literature.
First, the age of onset and the localization of the lesions are consistent with the classic epidemiological pattern of AD in infancy. According to Weidinger et al. (2018), in approximately 60% of cases the symptoms appear within the first year of life, with characteristic involvement of the cheeks, scalp, and extensor surfaces of the extremities. This localization pattern is partly explained by the greater exposure of these areas to mechanical irritants (contact with the pillow, friction during feeding, scratching) and by regional differences in the structure of the epidermal barrier (Eichenfield et al., 2014a).
The strongly positive family history of atopy in our case (maternal grandmother with atopic dermatitis, maternal uncle with asthma and allergic rhinitis) is consistent with the strongly hereditary nature of the disease. Large epidemiological studies have shown that the risk of developing AD is about 2–3 times higher in children with one atopic parent and up to 5 times higher when both parents are affected (Apfelbacher et al., 2011). FLG gene variants and other genes that regulate the epidermal barrier and the Th2 immune response are inherited along family lines and explain the familial clusters observed in clinical practice (Palmer et al., 2006; Irvine et al., 2011).
A critical aspect of the case is the presence of secondary infection with Staphylococcus aureus, confirmed by bacterial culture. S. aureus is well known as the most frequent pathogen in AD flares, with studies reporting colonization in up to 70–90% of active lesions (Geoghegan et al., 2018). The mechanisms by which S. aureus contributes to the pathogenesis of AD are numerous: production of superantigens such as the staphylococcal enterotoxins (SEA, SEB), which directly activate T lymphocytes without antigen specificity; production of alpha-toxin, which directly damages keratinocytes; and disruption of the epidermal barrier through the secretion of proteases (Mempel et al., 1998; Geoghegan et al., 2018). The honey-yellow crusts noted in our case represent a classic clinical sign of impetiginization of atopic lesions and require prompt treatment with antibiotics.
With regard to treatment, the approach followed reflects the best practices recommended in contemporary international guidelines (Eichenfield et al., 2014b; Wollenberg et al., 2018). At the heart of management lies the restoration of the epidermal barrier through frequent use of

emollients. Emollients containing ceramides and humectant substances contribute to restoring the intercellular lipids of the cutaneous barrier and to increasing skin hydration, thereby reducing transepidermal water loss and the intensity of pruritus. Application of the emollient within three minutes after bathing, a technique known as "soak and seal," is considered an evidence-supported strategy, since it favors optimal penetration of the preparation and the retention of moisture in the horny layer of the skin (Eichenfield et al., 2014; Cork et al., 2009; Sidbury et al., 2014).

The important study by Simpson et al. (2014) showed that daily application of emollients from birth in infants at high familial risk reduced the cumulative incidence of AD at 6 months of age by about 50%. Similar results were reported by Horimukai et al. (2014), who demonstrated a significant reduction of AD after the application of emollients from the neonatal period. These findings underscore the primary role of emollient care as a fundamental preventive and therapeutic measure, especially in infants with an at-risk family history — as in our case.

The choice of hydrocortisone 1% as a low-potency topical corticosteroid is reasonable and supported by the literature, especially in the context of the patient's young age and the localization of the lesions on the face. The skin of the face and intertriginous areas has greater permeability to corticosteroids and a higher risk of local side effects, including atrophy, telangiectasias, and perioral dermatitis. The use of high-potency corticosteroids in these areas and in infants should be regularly avoided. Although the frequent parental concern about the fear of using corticosteroids — often referred to as "corticophobia" — frequently leads to underuse and results in poor disease control (Charman et al., 2002), studies have shown that short-term, monitored use of low-potency corticosteroids is safe and highly effective (Hoare et al., 2000).

The use of topical mupirocin 2% represents an evidence-supported therapeutic strategy for the treatment of localized secondary infections with *Staphylococcus aureus* in patients with atopic dermatitis (AD). In the Cochrane review, Bath-Hextall et al. (2010) recommend the use of topical antibiotics for limited cutaneous infections, instead of systemic antibiotics, in order to reduce the risk of bacterial resistance and of systemic side effects. Mupirocin acts by inhibiting the bacterial enzyme isoleucyl-tRNA synthetase and is effective both against methicillin-sensitive *S. aureus* (MSSA) strains and against methicillin-resistant strains (MRSA), making it a rational first-line choice for localized bacterial infections in AD.

Parental education is considered a fundamental component in the long-term management of atopic dermatitis and has a direct impact on therapeutic success. Studies have shown that structured educational programs significantly improve the knowledge of caregivers, adherence to treatment, disease control, and the quality of life of patients and their families (Stalder et al., 2013; LeBovidge et al., 2016). In our case, the parents received detailed instructions regarding correct bathing and skin hydration practices, the proper use of topical corticosteroids, and the early recognition of warning signs of infection. This educational approach is thought to have contributed to the rapid clinical improvement of the patient.

An important long-term consideration is the "atopic march": the tendency for the later development of asthma, allergic rhinitis, and food allergies in children with AD that presents early and with increased severity (Spergel, 2010). Our patient, having AD with early onset and a strongly positive family history of atopy (including asthma in the maternal uncle), is at increased risk for this trajectory. Careful observation and early interventions may modify this course, although current data are still limited and further longitudinal observational studies are required.

Finally, it is important to emphasize the potential role of dietary factors. Although food allergy was not confirmed in our case (the infant was exclusively breastfed and presented no suggestive gastrointestinal or respiratory symptoms), in the group of patients with severe AD, and especially those refractory to standard treatment, evaluation for food allergies — particularly to cow's milk protein, eggs, and nuts — should be considered after consultation with the pediatric allergist (Eichenfield et al., 2014a; Boyce et al., 2010).
The limitations of this case presentation include its single-case nature and the relatively short period of follow-up (14 days). Nevertheless, the case offers a practical and didactic illustration of the management of AD in infancy with secondary infection, and it highlights basic principles that are valuable in everyday clinical practice.

Conclusion

Atopic dermatitis in infants remains an important clinical challenge, characterized by complex interactions among genetic, immunological, environmental, and microbial factors. The case presented of a 6-month-old infant with moderate-to-severe AD and secondary infection with Staphylococcus aureus illustrates several fundamental principles in the management of this disease in infancy.
Early diagnosis through careful clinical examination and recognition of the characteristic features in this age group; assessment of the family history of atopy as a prognostic indicator; early identification of secondary bacterial infections with microbiological confirmation for targeted treatment; an individualized therapeutic approach including restoration of the epidermal barrier with emollients, control of inflammation with topical corticosteroids of an appropriate potency for the patient's age and the lesion site, treatment of infections with topical antibiotics based on the antibiogram; and thorough parental education as an essential component of long-term management — together represent a successful treatment model.
The rapid and substantial clinical improvement of the patient after 14 days of treatment confirms the efficacy of the approach taken. However, given the chronic and relapsing nature of the disease, long-term follow-up remains essential. The identification of children at high familial risk of atopy may allow early preventive interventions with emollients, while future research may shed more light on the role of the skin microbiome, on biomarkers for patient stratification, and on new molecularly targeted therapies — such as JAK inhibitors and anti-IL-4/IL-13 monoclonal antibodies — which are opening new perspectives in the management of AD in older age groups and potentially also in infants.
In conclusion, this case underscores that the effective management of atopic dermatitis requires a comprehensive, individualized, and educational approach that takes into consideration the biological characteristics of the patient, the family context, and the evidence-based principles of contemporary literature.

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