Our Dermatology Online

Original Article

Atopic dermatitis in Senegalese children with skin phototype VI: Prevalence, clinical features, and risk factors of severity

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ABSTRACT

Background: Atopic dermatitis (AD) is the most common inflammatory skin disease of childhood. Yet, in sub-Saharan Africa, data on AD in children is scarce. Herein, we aimed to determine the prevalence, clinical features, and risk factors of severity of AD in Senegalese children with skin phototype VI. **Materials and Methods:** This was a cross-sectional study including children with AD and skin phototype VI younger than fifteen years old seen in two dermatology centers in Senegal over a period of six months. The diagnosis of AD was based on the United Kingdom Working Party (UKWP) criteria. The severity of AD was evaluated with SCORing of Atopic Dermatitis (SCORAD). **Results:** Among the 630 children consulted during the study period, 104 had AD, yielding a hospital prevalence of 16.5%. The mean age of children with AD was 36 months with a sex ratio of 1. A personal and family history of atopic disease was reported in 86.5% and 84.6% of the patients, respectively. Xerosis was the most common clinical feature, observed in 80.8% of. Post-inflammatory hyperpigmentation and keratosis pilaris were observed in 44.2% and 37.5%, respectively. Severe AD was noted in 12.5%. Risk factors associated with the severity of AD were exposure to incense smoke, an age of onset before 24 months, food allergies, and *impetiginisatio*. Daily use of shea butter was a protective factor. **Conclusion:** Our study showed a high hospital prevalence of AD in Senegalese children with skin phototype VI. The result observed with shea butter as a protective factor against severe AD is highly important, although it needs to be confirmed by randomized studies.

Key words: Atopic dermatitis; Children; Phototype VI; Senegal

INTRODUCTION

Atopic dermatitis (AD) is a common, highly pruritic chronic inflammatory skin disorder in children, which is often associated with other atopic diseases, such as asthma and allergic rhinitis [1].

The prevalence of AD varies significantly across the world [2]. Historically, the prevalence of AD in Africa has generally been lower than in the West, yet recent trends have shown an increasing prevalence in developing countries [3,4].

AD, especially in children, remains a particularly underestimated disease in sub-Saharan Africa, with a considerable lack of data in the general population and at the hospital level. Yet, it has become a real worldwide health problem due to its increasing prevalence, its negative impact on quality of life, and the high cost of its management [5]. In addition, in individuals with skin phototype VI, AD has numerous clinical specificities mainly linked to skin pigmentation [6]. Skin typing refers to the classification of the skin according to its sensitivity to sun exposure. The Fitzpatrick classification is the most widely employed

How to cite this article: Seck B, Diallo M, Ndiaye MT, Diop A, Ba ID, Ly F. Atopic dermatitis in Senegalese children with skin phototype VI: Prevalence, clinical features, and risk factors of severity. Our Dermatol Online. 2022;13(4):359-364.

Submission: 11.04.2022; **Acceptance:** 27.08.2022

DOI: 10.7241/ourd.20224.3

method of skin typing. According to this classification, skin phototype VI corresponds to black skin that never burns and always tans darkly [7].

Herein, we aimed to determine the prevalence, clinical features, and risk factors of severity of AD in Senegalese children with skin phototype VI.

MATERIALS AND METHODS

This cross-sectional, descriptive, and analytical study was conducted over a period of six months—from January 1 to July 1, 2019—in two dermatology centers in Senegal (Institute of Social Hygiene Hospital of Dakar and Regional Hospital Centre of Saint-Louis). The study population was children aged fifteen years or younger consulting for AD during the study period. For this study, the UKWP criteria for the diagnosis of AD were employed to identify the cases. A diagnosis of AD was reached in the presence of a pruritic rash and three or more of the following features:

- a history of rash in the skin creases (fold of the elbow, behind the knees, front of the ankles, and around the neck);
- a personal or family history of asthma and hay fever;
- history of generalized dry skin (xerosis);
- onset before the age of two years;
- visible flexural dermatitis.

Data was collected with a pre-established questionnaire. The clinical examination of the children was performed in the compulsory presence of the parent/guardian. The first step was an interrogation with the collection of sociodemographic data, the child's personal and family history, cosmetic habits, and anamnestic data. The child's physical examination was then conducted to identify the elementary lesions and their topography.

The SCORAD index was employed to assess the disease severity of AD. It consisted of a combination of three items: the extent of skin involvement, the intensity of dermatitis, and the subjective symptoms. To measure the extent of AD, the Wallace rule of nines was applied to the front/back drawing of the patient's inflammatory lesions. The extent was graded from 0 to 100. The intensity part of SCORAD consisted of six items: erythema, edema/papulation, excoriations, lichenification, oozing/crusts, and dryness. Each item was graded on a scale from 0 to 3. The subjective items included daily pruritus and sleeplessness. The SCORAD formula was as follows: A/5 + 7B/2 + C. Here, *A* was defined as the extent (0–100), *B* as the intensity (0–18), and *C* as the subjective symptoms (0–20). The maximal SCORAD score was 103. AD was classified as mild with SCORAD < 25, moderate with 25–50, and severe with > 50.

Data was entered into Microsoft Excel and analyzed with SPSS Statistics, version 26.0. The Pearson's chi-square test and the Fischer's exact test were employed to find an association between the dependent variable (presence of severe AD) and the covariables, with the significance threshold at $p < 0.05$. In the case of a significant association, we assessed the strength of this association with the odds ratio with 95% confidence limits.

The participants and/or their parents/guardians were provided with detailed information on the study and assured that confidentiality would be ensured. Informed consent from the parent and/or guardian was required before inclusion.

RESULTS

A total of 630 children with skin diseases were seen during the study period. Among these, 104 had AD according to the UKWP diagnostic criteria, giving a hospital prevalence of 16.5%. Their mean age was 56 months and the sex ratio was 1. Among the children with AD, 29 (27.9%) were infants and 75 (72.2%) were children older than two years.

A personal history of the atopic disease was reported in 90 (86.5%) patients. These were allergic rhinitis in 66 (63.5%), asthma in 36 (34.6%), allergic conjunctivitis in 28 (26.9%), and a food allergy in 13 (12.5%) cases. AD was the first step in the "atopic march" in 61 (58.7%) patients. A family history of atopy in the first degree was reported in 88 (84.6%) patients.

Thirty-six (34.6%) patients developed AD within the first six months of life, 64 (61.5%) before the age of two years, and 93 (89.4%) before the age of five years.

The mean duration of AD was 26.5 days, with extremes ranging from five to ninety days. Pruritus was present in all patients and sleeplessness in 47 (45.2%).

The main clinical feature was xerosis, observed in 84 (80.8%) patients. The erythematous-squamous

aspect, corresponding to chronic eczema, was observed in 82 (78.8%). Some minor signs of AD such as post-inflammatory hyperpigmentation and keratosis pilaris were also common, observed in 46 (44.2%) and 39 (37.5%) patients, respectively. *Impetiginisatio* of the lesions was observed in 26 (25%) patients. Table 1 shows the distribution of the different clinical features of AD observed in our patients depending on age.

The most frequent locations of the eczematic lesions were the trunk in 72 (69.2%) cases, the face in 49 (47.4%), the folds of the elbow in 50 (48.1%), the neck in 46 (44.2%), and the knee in 44 (42.3%). Table 2 shows the distribution of the different topographies in our patients depending on age.

The mean SCORAD score was 31.3. AD was mild in 35.6% (n = 37), moderate in 51.9% (n = 54), and severe in 12.5% (n = 13). The risk factors associated with the severity of AD were regular exposure to incense smoke (p = 0.009; OR = 6.4 [1.9–17.4]), a food allergy (p < 0.001; OR = 10.3 [2.7–39.1]), an age of onset before twenty-four months (p = 0.01; OR = 9 [1.2–13.7]) and *impetiginisatio* (p = 0.01; OR= 4.4 [1.3–4.7]). Daily application of shea butter to the entire body was a protective factor (p = 0.005; OR = 0.1 [0.01–0.7]) (Table 3).

Table 1: Distribution of the different clinical features of AD in our patients depending on age.

Clinical Feature	Infants	Age≥2 yrs.	Total
	n (%)	*n* (%)	*n* (%)
Xerosis	18 (62.1)	66 (88)	84 (80.8)
Erythematous-squamous aspect	21 (72.4)	61 (81.3)	82 (78.8)
Post-inflammatory hyperpigmentation	7 (24.1)	39 (52)	46 (44.2)
Keratosis pilaris	8 (27.6)	31 (41.3)	39 (37.5)
Excoriation/ulceration	6 (20.7)	21 (28)	27 (25.9)
Lichenification	2 (7)	23 (30.7)	25 (24)
Retro/infra-auricular fissures	1 (3.4)	18 (24)	19 (18.3)
Dennie–Morgan lines	2 (6.9)	9 (12)	11 (10.6)
Acute eczema lesions	3 (10.3)	7 (9.3)	10 (9.6)
Prurigo	none	9 (12)	9 (8.7)
Palmoplantar keratoderma	1 (3.4)	6 (8)	7 (6.7)
Pityriasis alba	none	5 (6.7)	5 (4.8)
Nummular dermatitis	1 (3.4)	3 (4)	4 (3.8)
Erythroderma	1 (3.4)	1 (2.7)	2 (1.9)

Table 2: Distribution of the different topographies in our patients according to age

Location	Infants	Age≥2 yrs.	Total
	n (%)	*n* (%)	*n* (%)
Trunk	18 (62.1)	54 (72)	72 (69.2)
Face	18 (62.1)	31 (41.3)	49 (47.4)
Extensions of the limbs	14 (48.3)	26 (34.7)	40 (38.5)
Folds			
Elbows	10 (34.5)	40 (53.3)	50 (48.1)
Neck	16 (55.2)	30 (40)	46 (44.2)
Knees	9 (31)	35 (46.7)	44 (42.3)
Armpits	4 (13.8)	13 (17.3)	17 (16.3)
Groin	3 (10.3)	3 (4)	6 (5.8)
Scalp	13 (44.8)	20 (26.7)	33 (31.7)
Buttocks	5 (17.2)	11 (14.7)	16 (15.4)
External genitals	6 (20.7)	6 (8)	12 (11.5)
Periorbital rims	none	9 (12)	9 (8.7)
Ears	none	6 (8)	6 (5.8)

DISCUSSION

The hospital prevalence of AD in children with skin phototype VI is variously assessed in sub-African series. The prevalence of 16.5%, found in our study, differs from that reported by Técléssou et al. in Togo with 31.3% and from that reported by Ahogo et al. in Ivory Coast with 9.2% [8,9]. The differences in prevalence also exist in a Chinese series, with the rates ranging from 9% to 24% [10]. These differences in the prevalence of AD are mainly due to difficulties in interpreting the criteria employed in the diagnosis of AD, especially those proposed by the UKWP. The differences in methodology are also sometimes noted, as in the study by Técléssou, in which recruitment was exclusively in vaccination centers [8]. In any case, the prevalence of AD remains high in African hospital studies, making AD an emerging disease in sub-Saharan Africa.

Air pollution also plays a key role in the prevalence and severity of AD [11]. Our study found a higher risk of severe AD in children regularly exposed to incense smoke. There are some studies on the effect of incense smoke on AD. In China, ZHANG et al. found a significant association between exposure to incense and increased prevalence of AD in children aged one to eight years [12]. To our knowledge, no studies have correlated the severity of AD with exposure to incense smoke. However, chemical analysis of incense smoke reveals a complex mixture of particulate and gaseous pollutants that may aggravate AD. Incense burning, which is an incomplete combustion process, is known to emit fine and ultrafine particles ($PM_{2.5}$ and $PM_{0.1}$) in large quantities, carbon monoxide (CO), nitrogen oxides (NO_x), toxic polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds (VOCs), such as benzene and isoprene [13,14]. The effects of fine and ultrafine particles on the severity of AD were investigated in a longitudinal study by Song

Table 3: Risk factors associated with severe atopic dermatitis (univariate analysis).

Characteristics	Severe Atopic Dermatitis		*p* Value	OR [95% CI]
	Present n (%)	Absent n (%)		
Sex				
male	7 (13.5)	45 (86.5)	0.38	
female	6 (11.5)	46 (88.5)		
Age group				
infant	3 (10.3)	26 (89.7)	0.48	
children over 2 yrs.	10 (13.3)	65 (86.7)		
Exclusive breastfeeding	6 (9.8)	55 (90.2)	0.14	
Weaning age before the 18 month	2 (10.5)	17 (89.5)	0.47	
Food introduction before 6 months	4 (23.5)	13 (76.5)	0.17	
Incense exposure	11 (20.8)	42 (79.2)	0.009	6.4 [1.9–17.4]
Use of scented toiletries	8 (19)	34 (81)	0.06	
Daily use of shea butter	1 (2.3)	43 (97.7)	0.005	0.1 [0.01–0.7]
Asthma	7 (19.4)	29 (80.6)	0.07	
Allergic rhinitis	9 (13.6)	57 (86.4)	0.44	
Allergic conjunctivitis	3 (10.7)	25 (89.3)	0.51	
Food allergy	6 (46.2)	7 (53.8)	< 0.001	10.3 [2.7–39.1]
Age of onset before 24 months	12 (18.8)	52 (81.2)	0.01	9 [1.2–13.7]
Impetiginisatio	7 (26.9)	19 (73.1)	0.01	4.4 [1.3–4.7]

et al., which included 41 schoolchildren aged eight to twelve years. The study found that the itching score was significantly associated with the concentrations of ambient fine and ultrafine particles [15]. Concerning the effect of gaseous pollutants, a nationwide survey of middle school students in Taiwan involving 317,926 children demonstrated that severe flexural eczema was positively associated with exposure to CO and NO_x [16]. A double-blind, cross-over study by Huss-Marp et al. demonstrated that VOCs increased transepidermal water loss (TEWL) in patients with AD 48 hours after exposure, which aggravates xerosis [17]. Given all these findings, the aggravation of AD after exposure to incense smoke is easy to understand.

Regarding cosmetic habits, shea butter was found to be a protective factor against severe AD in our study. The protective effect of shea butter is explained by its anti-inflammatory and moisturizing properties. The anti-inflammatory properties of shea butter are attributed to the several derivatives of cinnamic acid contained therein [18]. Shea butter also contains vitamins A and E and has semi-solid characteristics and buttery consistency, which makes it an effective emollient and moisturizer for the skin [19]. Thus, shea butter could be an excellent alternative to classic emollients, which are often inaccessible to the majority of patients.

In our study, AD was frequently associated with a personal or family history of atopy. The frequent association between AD and a personal or family history of atopy is well documented in the literature [20,21]. Regarding the relationship between atopic diseases and the severity of AD, only a food allergy was statistically associated with severe AD in our study. This association was reported by several authors. In Turkey, Celiksoy et al. showed that sensitization to food allergens such as cow's milk and hen's eggs was significantly associated with the severity of AD in young children [22].

Xerosis was the most frequent clinical feature in our patients, observed in 80.8% of the cases. In a study by Bayonne-Kombo et al. from Congo, xerosis was also the most frequent feature, in 89.33% of the cases [23]. A meta-analysis by Yew et al. revealed that xerosis was the most frequent aspect of AD in all regions of the world, except southeast Asia [24]. The meta-analysis reported an average frequency of 65% for xerosis in three African regions: Nigeria, Tunisia, and South Africa [24]. The frequency of xerosis in atopic patients is explained by increased TELW, abnormalities in essential fatty acid metabolism, a decrease in ceramides in the stratum corneum, and a decrease in the content of hygroscopic molecules and their precursors.

Some minor signs of AD, such as keratosis pilaris and retroauricular fissures, were relatively common in our series. Keratosis pilaris was observed in 37.5% of our patients. Its frequency was 38.8% in an Ivorian study by Ahogo [9]. Compared to other

phototypes, keratosis pilaris appears more frequently in phototype VI according to several reviews [25]. Individuals with phototype VI also have a higher risk of developing pigment disorders after inflammatory dermatosis [26]. In our study, 42.2% of the patients had post-inflammatory hyperpigmentation. Depending on its extent, it may have a considerable impact on the child's self-esteem and quality of life.

In our study, *impetiginisatio* was common and associated with the risk of severe AD. Patients with AD have an increased risk of bacterial skin infections [27]. Staphylococcus aureus colonizes the skin of most patients with AD and is the most common organism to cause infections [28]. The correlation between bacterial colonization and the severity of eczema was reported by several studies in the literature [29].

CONCLUSION

Our study revealed a high hospital prevalence of AD in Senegalese children with skin phototype VI. The frequency of xerosis, post-inflammatory hyperpigmentation, and keratosis pilaris remains the main clinical characteristics of AD in children with skin phototype VI. Our study was remarkable for the contribution of shea butter in reducing the severity of AD. This result is promising and should be confirmed by randomized studies on large numbers of patients. The parapharmaceutical emollients classically employed for this purpose are often inaccessible in our region because of their high cost.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Frazier W, Bhardwaj N. Atopic dermatitis: Diagnosis and treatment. Am Fam Physician. 2020;101:590-8.
2. Deckers IA, McLean S, Linssen S, Mommers M, van Schayck CP, Sheikh A. Investigating international time trends in the incidence and prevalence of atopic eczema 1990-2010: A systematic review of epidemiological studies. PLoS One. 2012;7:e39803.
3. Weidinger S, Novak N. Atopic dermatitis. Lancet. 2016; 387:1109-22.
4. Mallol J, Crane J, von Mutius E, Odhiambo J, Keil U, Stewart A; ISAAC Phase Three Study Group. The International Study of Asthma and Allergies in Childhood (ISAAC) Phase Three: A global synthesis. Allergol Immunopathol (Madr). 2013;41:73-85.
5. Nutten S. Atopic dermatitis: Global epidemiology and risk factors. Ann Nutr Metab. 2015;66 Suppl 1:8-16.
6. Al-Afif KAM, Buraik MA, Buddenkotte J, Mounir M, Gerber R, Ahmed HM, et al. Understanding the burden of atopic dermatitis in Africa and the Middle East. Dermatol Ther (Heidelb). 2019;9:223-41.
7. Gupta V, Sharma VK. Skin typing: Fitzpatrick grading and others. Clin Dermatol. 2019;37:430-6.
8. Técléssou JN, Mouhari-Toure A, Akakpo S, Bayaki S, Boukari OBT, et al. [Risk factors and allergic manifestations associated with atopic dermatitis in Lomé (Togo): A multicenter study of 476 children aged 0-15 years]. Med Sante Trop. 2016;26:88-91.
9. Ahogo KC, Kouassi YI, Gbery IP, Azagoh KR, Yeboua KI, Kouassi KA, et al. Atopic dermatitis in children: Epidemiological and clinical aspects in Côte d'Ivoire. Our Dermatol Online. 2017;8(Suppl.1):25-7.
10. Guo Y, Li P, Tang J, Han X, Zou X, Xu G, et al. Prevalence of atopic dermatitis in Chinese children aged 1-7 ys. Sci Rep. 2016;6:29751.
11. Ahn K. The role of air pollutants in atopic dermatitis. J Allergy Clin Immunol. 2014; 134:993-9.
12. Zhang M, Wu Y, Yuan Y, Zhou E, Sundell J, Sun Y, et al. Effects of home environment and lifestyles on prevalence of atopic eczema among children in Wuhan area of China. Chinese Science Bulletin. 2013;58:4217-22.
13. Lin TC, Krishnaswamy G, Chi DS. Incense smoke: clinical, structural and molecular effects on airway disease. Clin Mol Allergy. 2008;6:3.
14. Ji X, Le Bihan O, Ramalho O, Mandin C, D'Anna B, Martinon L, et al. Characterization of particles emitted by incense burning in an experimental house. Indoor Air. 2010;20:147-58.
15. Song S, Lee K, Lee YM, Lee JH, Lee SI, Yu SD, et al. Acute health effects of urban fine and ultrafine particles on children with atopic dermatitis. Environ Res. 2011;111:394-9.
16. Lee YL, Su HJ, Sheu HM, Yu HS, Guo YL. Traffic-related air pollution, climate, and prevalence of eczema in Taiwanese school children. J Invest Dermatol. 2008;128:2412-20.
17. Huss-Marp J, Eberlein-König B, Breuer K, Mair S, Ansel A, Darsow U, et al. Influence of short-term exposure to airborne Der p 1 and volatile organic compounds on skin barrier function and dermal blood flow in patients with atopic eczema and healthy individuals. Clin Exp Allergy. 2006;36:338-45.
18. Maanikuu PM, Peker K. Medicinal and nutritional benefits from the shea tree (*Vitellaria paradoxa*). J Biol Agric Healthcare. 2017;7:51-7.
19. Ayanlowo O, Ebie C, Cole-Adeife O, Ilomuanya M, Adegbulu A. Shea butter as skin, scalp, and hair moisturizer in Nigerians. Dermatol Ther. 2021;34:e14863.
20. Gallay C, Meylan P, Mermoud S, Johannsen A, Lang C, Rivolta C, et al. Genetic predisposition and environmental factors associated with the development of atopic dermatitis in infancy: A prospective birth cohort study. Eur J Pediatr. 2020;179:1367-77.
21. Thomsen SF. Epidemiology and natural history of atopic diseases. Eur Clin Respir J. 2015;2.
22. Celiksoy MH, Topal E, Demir O. Age-related risk factors associated with severity of atopic dermatitis in children. Ann Med Res. 2019;26:165-8.
23. Bayonne-Kombo ES, Loubove H, Mavoungou YGV, Gathsé A. Clinical aspects of atopic dermatitis of children in Brazzaville, Congo. Open Dermatol J. 2019;13:61-7.
24. Yew YW, Thyssen JP, Silverberg JI. A systematic review and meta-analysis of the regional and age-related differences in

atopic dermatitis clinical characteristics. J Am Acad Dermatol. 2019;80:390-401.
25. Kaufman BP, Guttman-Yassky E, Alexis AF. Atopic dermatitis in diverse racial and ethnic groups: Variations in epidemiology, genetics, clinical presentation and treatment. Exp Dermatol. 2018;27:340-57.
26. Alexis AF, Blackcloud P. Psoriasis in skin of color: Epidemiology, genetics, clinical presentation, and treatment nuances. J Clin Aesthet Dermatol. 2014;7:16-24.
27. Langan SM, Abuabara K, Henrickson SE, Hoffstad O, Margolis DJ. Increased risk of cutaneous and systemic infections in atopic dermatitis: A cohort study. J Invest Dermatol. 2017;137:1375-7.
28. Altunbulakli C, Reiger M, Neumann A U, Garzorz-Stark N, Fleming M, Huelpuesch C, et al. Relations between epidermal barrier dysregulation and Staphylococcus species–dominated microbiome dysbiosis in patients with atopic dermatitis. J Allergy Clin Immunol. 2018;142:1643-7.
29. Petry V, Lipnharski C, Bessa GR, Silveira VB, Weber MB, Bonamigo RR, et al. Prevalence of community-acquired methicillin-resistant Staphylococcus aureus and antibiotic resistance in patients with atopic dermatitis in Porto Alegre, Brazil. Int J Dermatol. 2014;53:731-5.

Source of Support: Nil, **Conflict of Interest:** The authors state no conflict of interest.