THE MEDICAL BULLETIN OF
SISLI ETFAL HOSPITAL

DOI: 10.14744/SEMB.2018.67365
Med Bull Sisli Etfal Hosp 2019;53(1):58–69

Original Research

Prevalence of Atopic Dermatitis Criteria among Textile Workers with Occupational Allergic Contact Dermatitis and Effects of Having Atopic Dermatitis on Contact Antigenic Diversity

Betül Taş,[1] İlknur Kıvanç Altunay[2]

[1]Department of Dermatology and Venereology, University of Health Sciences, Istanbul Bagcilar Training and Research Hospital, Istanbul, Turkey
[2]Department of Dermatology and Venereology, University of Health Sciences, Sisli Hamidiye Etfal Teaching and Research Hospital, Istanbul, Turkey

Abstract

Objectives: Contact dermatitis (CD) is a common skin disease. Occupational contact dermatitis (OCD) is the most frequently seen occupational skin disease and includes both occupational allergic CD (OACD) and occupational irritant CD (OICD). One of the most common sources of OACD is textile products. Individuals with atopic dermatitis (AD) have an increased risk for development of allergic contact dermatitis (ACD). However, the role of AD in the etiopathogenesis of the development of OACD among textile industry workers is not well known. The aim of the present study was to determine the prevalence of AD among textile workers with OACD and to analyze contact antigenic diversity between the workers with and without AD.

Methods: A prospective, cross-sectional study was conducted with 352 textile workers who had previously been diagnosed with OACD. The patients were questioned and examined with respect to AD criteria, demographic features, disease duration, duration of employment until first symptoms, phototype, workplace (subsectors), and location of lesions at control visits. Immediate skin test reactivity was evaluated with a commercial skin prick test panel. The data obtained and the patients' previously recorded patch test results were compared in OACD groups with and without a diagnosis of AD. The results were statistically evaluated with a significance level of p value <0.05.

Results: The study population consisted of 124 males and 227 females. The mean age was 35.69±13.65 years. The most commonly seen employment duration, phototype, subsector, and location were 4 to 8 months (26.14%), 9 to 12 months (34.66%), Fitzpatrick type-III (37.50%), dyeing (33.52%), and exclusively the hands (60.51%), respectively. In all, 193 patients (54.83%) met the criteria for the diagnosis of AD. In the OACD group with AD, there was a significant number with 4 major and 16 minor criteria, as well as positivity for 14 contact allergens.

Conclusion: Most AD criteria, or a diagnosis of AD, are highly detectable among workers with textile-related OACD. The results for patch test allergens may be significantly higher than those of individuals without AD. Textile workers with AD should be warned about the possibility of the early development of OACD.

Keywords: Allergic contact dermatitis; antigenic diversity; atopic dermatitis; contact hypersensitivity; occupational dermatitis; textile.

Please cite this article as "Taş B, Kıvanç Altunay İ. Prevalence of Atopic Dermatitis Criteria among Textile Workers with Occupational Allergic Contact Dermatitis and Effects of Having Atopic Dermatitis on Contact Antigenic Diversity. Med Bull Sisli Etfal Hosp 2019;53(1):58–69".

Address for correspondence: Betül Taş, MD. Bagcilar Egitim ve Arastirma Hastanesi, Saglik Bilimleri Universitesi, Dermatoloji ve Venereoloji Anabilim Dali, İstanbul, Turkey
Phone: +90 533 432 95 46 **E-mail:** betulavc@yahoo.com

Submitted Date: July 11, 2018 **Accepted Date:** August 16, 2018 **Available Online Date:** February 04, 2019

Allergic contact dermatitis (ACD) and atopic dermatitis (AD) are common and burdensome cutaneous disorders that may present with similar signs and symptoms, such as pruritus, a burning sensation, scaling, erythema, and indurated plaques.[1] Although a definitive relationship between the 2 diseases is controversial, some reports suggest that individuals with AD have an increased likelihood of contact hypersensitivity.[2,3]

Research has indicated that patients with OACD reveal a 60% prevalence of atopy, compared with 30% in the general population.[4] Moreover, it has been stated that ACD patients with AD are more likely to have positive patch test results compared with patients without AD.[5] Each disease reflects distinct cutaneous sensitization to certain allergens and epidermal barrier dysfunction.[4,5] In principle, ACD is caused by Type IV hypersensitivity alone, whereas AD is a complex inflammatory process with Type I and IV allergies present in a complicated form. Although the majority of AD patients have atopic diathesis, this condition may not be valid for all AD patients.[6]

There is increased transcutaneous penetration of allergens and increased barrier dysfunction in those with AD, often due to increased use of topical products. It has been suggested that these factors may lead to potential antigen sensitization and presentation, and may therefore predispose an individual with AD to developing ACD.[7]

Although certain determinants, such as moisture, friction, sweat, airborne particles, genetic predisposition, and atopy, are known to be facilitating factors for the development of OACD,[8] there is no report regarding the prevalence of AD criteria in patients with textile OACD or contact antigenic diversity between OACD patients with and without AD. This study was conducted with the objective of adding to knowledge of the subject.

Methods

A prospective, cross-sectional study was conducted with 352 textile employees from 63 small-to-medium sized factories located in the Bagcilar district of Istanbul, Turkey. It was approved by the local ethics committee. The participants were randomly selected from individuals who had previously presented at the dermatology outpatient clinic, had been diagnosed with OACD with a patch test, and who were still in follow-up.

Written, informed consent was obtained from each member of the study before inclusion. The patients were examined and questioned by 2 dermatologists during control visits occurring between January and June 2018.

The inclusion criteria were volunteering for the study, providing the required written consent, possessing the ability to understand the questions, age greater than 18 years, work for at least 3 months at a textile manufacturing site, the presence for at least 1 month of dermatitis which had previously been diagnosed as OACD with a patch test, and the presence of lesions clinically compatible with ACD.

Pregnant women; patients who received any systemic or topical treatment that might affect the clinical condition of the lesions and skin prick test results, including antihistamines, corticosteroids, bronchodilators, mast cell stabilizers, H2-receptor agonists, tricyclic antidepressants, or immunosuppressive drugs in the previous 2 weeks; those who were known to be have any current immunodeficiency status, such as malignancy or HIV infection; and those with any condition that might lead to changes in their immune responses, such as diabetes, thyroid or renal dysfunction, or autoimmune diseases; and individuals who had a serious, active bacterial or viral infection were excluded from the study.

A previously prepared questionnaire was administered to the study participants to determine demographic features (age, gender, and marital status), disease duration (1-4, 4-8, 8-12, 12-16, and >16 months), duration of current employment until the first appearance of OACD symptoms (3-6, 6-9, 9-12, and >12 months), and skin phototype (I-V). Textile manufacturing subsectors of employment were categorized in 6 different areas: accessory application, dyeing, sewing, cutting, knitting, and packing. Lesions were diagnosed with dermatological examination findings, such as erythema, papules, vesicles, scaling, fissures, spread of lesions, and subjective symptoms. The location of lesions was recorded as the hands, hands/forearms, hands/face, or hands/trunk, according to the region where they were most concentrated.

Routine laboratory tests were performed, including a hemogram, blood biochemistry, and measuring the level of immunoglobulin E (IgE). Subsequently, the patients were surveyed using a questionnaire of AD criteria developed by Hanifin and Rajka in 1980. According to these criteria, the diagnosis of AD requires the presence of at least 3 major and 3 minor criteria.[6] During this processes, any unfamiliar medical terms were explained to the participants to ensure their understanding. The patient responses, laboratory test results, and examination findings of the physicians were used to determine the study data related to personal/family histories, subjective symptoms, clinical properties, and findings associated with AD.

Immediate (Type I) skin test reactivity was checked with a commercial skin prick test (SPT) panel (ALK-Abelló, Hørsholm, Denmark). A total of 38 allergens with standard activity and concentration were tested on the palmar surface

of the forearms (at least 5 cm from the wrist and 3 cm from the elbow): Dermatophagoides mix (D. pteronyssinus, D. farina), latex, cow's milk, Juglans regia, Spinacia oleracea, Fragaria vesca, Citrus x sinensis, Arachis hypogaea, Prunus persica, Theobroma cacao, Musa x sapientum, gliadin, fish mix I (sea bream, anchovy, red mullet, sardine), fish mix II (codfish, sole, sea bass, hake), Pullus gallinaceus, wasp, honeybee, Blattella germanica, Canis familiaris, Felis domesticus, feather mix, egg white and yolk, wheat, barley, oat and rye flours, pollens III (Avena sativa, Hordeum vulgare, Triticum sativum, Secale cereale), pollens IV (Dactylis glomerata, Festuca pratensis, Lolium perenne, Phleum pratense, Poa pratensis), Urtica dioica, Artemisia vulgaris, tree mix (Betula verrucosa, Corylus avellana, Olea europaea, Quercus ilex, Robinia pseudoacacia), Alnus glutinosa, Pinus sylvestris, Platanus x acerifolia, Populus nigra, Alternaria alternata, Cladosporium mix (C. fulvum, C. herbar). A negative control solution (with sodium chloride 0.9% solution) and a positive control fluid (with histamine hydrochloride 0.1%) were used according to the routine procedure.

Drops of the allergen extracts were placed on marked areas of the skin 2 cm apart. Using sterile lancets, small pricks were made vertically through the drops. The skin response to all of the allergens and both controls was interpreted 20 minutes after the application. The results were evaluated according to the guidelines of the European Academy of Allergy and Clinical Immunology, in which a positive result is defined as a wheal ≥3 mm diameter compared with the negative control. A positive SPT reaction to at least 1 of the allergen extracts was accepted as the presence of sensitization in that patient.[9]

Total serum IgE was measured using the photometric method and a commercial total human IgE test kit (Biomed Labordiagnostik GmbH, Oberschleißheim, Germany) with an automated analyzer (AU5840; Beckman Coulter Inc., Brea, CA, USA), using the measurement range of 10-1000 IU/mL (reference threshold: 100 IU/mL).

Previously performed patch test results of the study group patients were obtained from the medical files of the hospital database and used to compare the antigenic diversity of OACD subjects with and without AD. In our clinic, the Thin-layer Rapid-Use Epicutaneous test (TRUE test; Smartpractice ApS, Hillerød, Denmark) is used to determine lateonset hypersensitivity in patients who are thought to have ACD. The provocation test includes 3 adhesive panels with different allergen patches that are applied on the back 2 times at 2-day intervals. The results are read at day 2 and day 4 and interpreted according to the guidelines of the International Contact Dermatitis Research Group as (-), (+), (++), or (+++).[10]

Confirmation of an OACD diagnosis is based on the following criteria: (i) clinical confirmation of OCD, (ii) exposure to suspected occupational allergen(s), (iii) confirmation of a positive response to at least 1 of the relevant occupational patch test allergens, and (iv) confirmation of exposure as a cause or as an important aggravating factor in the development of the lesion. The population of the present study comprised participants who had had at least a (++) response to at least 1 of the allergens, since a (+) response can be interpreted as suspicious positivity.[11] The study was conducted in accordance with the Declaration of Helsinki.

Statistical Analysis

The statistical analysis was performed using NCSS 2007 software (NCSS LLC, Kaysville, UT, USA). Standard descriptive statistics were expressed as number (n) and mean±SD. Categorical variables were expressed as percentages (%). Chi-square or Fisher exact tests were used to compare qualitative data according to sample size in each OACD groups. The significance of differences in mean was determined using 95% confidence intervals, and a p value <0.05 was considered statistically significant.

Results

A total of 352 patients with OACD, 124 males (35.33%) and 227 females (64.67%), were included in the study. Demographic details, disease and employment duration, skin phototype, workplace subsector, and the location of the lesions in the study population are presented in Table 1. In the entire study group, the minimum and maximum age, mean age, and age group with the greatest incidence was 18 and 88 years, 35.69±13.65 years, and 18-30 years (42.05%), respectively.

A comparison of the demographic details, disease and employment duration, skin phototype, workplace subsector, and the location of the lesions according to groups selected based on a diagnosis of AD are provided in Table 2. There were no significant differences between the groups in terms of demographic features. Disease duration of 8-12, 12-16, and >16 months, and phototypes I and II were statistically more prevalent in the group with AD, whereas the length of employment until the first appearance of symptoms was lower in the OACD group with AD: 9-12 and >12 months (each p=0.0001). The accessory, dyeing, and knitting subsectors were statistically more frequently represented in the AD group (each p=0.0001). The incidence of lesions in the locations of hands/face, hands/forearms, and hands/trunk locations was greater in the OACD group with AD (each p=0.0001).

Table 1. Demographics, disease and employment duration, skin phototype, workers' subsector, and location of the lesions of the study group

Variables	Total group	
	n	%
Age (years)		
18-30	148	42.05
31-43	118	33.52
>44	86	24.43
Gender		
Male	124	35.33
Female	227	64.67
Marital status		
Married	206	58.52
Single	146	41.48
Disease duration (months)		
1-4	51	14.49
4-8	92	26.14
8-12	83	23.58
12-16	85	24.15
>16	41	11.65
Employment duration until first symptoms (months)		
3-6	74	21.02
6-9	78	22.16
9-12	122	34.66
>12	78	22.16
Phototype		
1	55	15.63
2	96	27.27
3	132	37.50
4	65	18.47
5	4	1.14
Subsector		
Accessory	62	17.61
Dyeing	118	33.52
Sewing	82	23.30
Cutting	40	11.36
Knitting	16	4.55
Packing	34	9.66
Atopic dermatitis		
Absent	159	45.17
Present	193	54.83

*AD: Atopic dermatitis.

A comparison of the distribution of the AD criteria of Hanifin and Rajka according to groups defined by an AD diagnosis is provided in Table 3. In all, 193 (54.83%) patients had at least 3 major and 3 minor criteria for AD. The presence of all major and 16 of 23 minor criteria was significantly higher in the OACD group with AD compared with the group without AD. Four (1.14%) allergic prick test responses qualified as positive, with a similar response in each group: Two (1.26%) grass mix reactions were seen in the group without AD, and 2 (1.04%) reactions were observed in the group with AD, 1 positive result for grass mix and 1 for house dust mites. No significant difference was found (p=0.845). The serum IgE level was determined to be above the threshold in 1 member of the group without AD (n=1, 0.63%), whereas it was elevated in 5 patients in the AD group (n=5, 2.59%). However, the difference was not significant (p=0.157). When the responses of the whole group to the patch test allergens were evaluated, the most notable sensitivities were detected for nickel sulphate (n=69, 19.60%), cobalt chloride (n=37, 10.51%), 13-p-tert-butylphenol formaldehyde resin (n=33, 9.38%) and epoxy resin (n=32, 9.09%). The paraben mix (n=9, 2.52%), colophony and methyldibromo glutaronitrile (each n=10, 2.84%), and neomycin sulphate (n=14, 3.98%) produced the least frequent responses.

Comparison of the allergen patch test results by group are shown in Table 4. Fourteen allergens showed significantly higher positivity in the OACD group with AD.

Discussion

The relationship between contact hypersensitivity and atopy is controversial. Some reports suggest that contact hypersensitivity is greater in individuals with AD, whereas others do not support the notion of this relationship. Although the available studies are as yet insufficient to pinpoint the exact relationship between AD and ACD, it is apparent that the 2 conditions may frequently coexist. Nonetheless, it is not yet clear whether this association is the result of coincidence or a common pathogenesis.[3-5,12]

Uehara et al.[13] reported that 33%, 100%, and 95% of patients with severe, mild, and moderate AD, respectively, demonstrated positive challenge test results to the potent contact sensitizer dinitrochlorobenzene, which indicated decreased contact sensitivity in severe AD. Conversely, Mailhol et al.[14] found that the severity of AD was a significant risk factor for developing contact hypersensitivity. Also, Czarnobilska et al.[15] reported that a concomitance of ACD and AD was observed in 33% of children and 73% of adolescents in their study. The prevalence of AD among patients with OACD was 54.83% in the present study group. Our result is consistent with previous studies regarding a positive relationship between the 2 condition. Moreover, all of the major and 16 minor AD criteria were found in significantly high numbers in the OACD group with AD in comparison with the group without AD.

An immediate skin test reaction was seen in only 2 participants in each group. An elevated serum IgE level was de-

Table 2. Comparison of demographics, disease and employment duration, skin phototype, workers'subsector, and location of lesions according to the OACD groups with and without AD

Subgroups of variables	OACD patients without atopic dermatitis (-)		OACD patients wiht atopic dermatitis (+)		p
	n=159	%	n=193	%	
Age (years)					
18-30	60	37.74	88	45.60	0.158
31-43	53	33.33	65	33.68	
>44	46	28.93	40	20.73	
Gender					
Male	61	38.61	63	32.64	0.245
Female	97	61.39	130	67.36	
Marital status					
Married	101	63.52	105	54.40	0.084
Single	58	36.48	88	45.60	
Disease duration (month)					
1-4	47	29.56	4	2.07	0.0001
4-8	61	38.36	31	16.06	
8-12	27	16.98	56	29.02	
12-16	16	10.06	69	35.75	
>16	8	5.03	33	17.10	
Employment duration until first symptoms (months)					
3-6	53	33.33	21	10.88	0.0001
6-9	41	25.79	37	19.17	
9-12	75	38.86	47	29.56	
>12	60	31.09	18	11.32	
Phototype					
1	13	8.18	42	21.76	0.0001
2	24	15.09	72	37.31	
3	63	39.62	69	35.75	
4	36	22.64	29	15.02	
5	3	1.89	1	0.52	
Subsector					
Accessory	19	11.95	43	22.28	0.0001
Dyeing	27	16.98	91	47.15	
Sewing	70	44.03	12	6.22	
Cutting	24	15.09	16	8.29	
Knitting	2	1.26	14	7.25	
Packing	17	10.69	17	8.81	
Location					
Hands	113	71.07	100	51.81	0.0001
Hands/forearms	38	23.90	60	31.09	
Hands/face	2	1.26	15	7.77	
Hands/trunk	6	3.77	18	9.33	

AD: Atopic dermatitis; OACD: Occupational allergic contact dermatitis.

termined in 5 individuals in the OACD group with AD, while there was only 1 case in the other group. All of the differences were insignificant. As we did not find a similar study in literature, it was not possible to make any comparison in this regard.

No racial predilection has been reported for ACD, it is more common in women than in men, and the incidence appears to increase with age.[16] Textile OACD cases, however, are more frequently reported in men and those who may only be in their thirties.[17-19] The majority of our patients

Table 3. Comparison of the distribution of AD* criteria of Hanefin & Rajka according to the OACD** groups

Atopic dermatitis criteria of Hanifin&Rajka	OACD patients without atopic dermatitis (-)		OACD patients with atopic dermatitis (+)		p
	n=159	%	n=193	%	
Major Criteria					
Pruritus					
Absent	51	32.08	0	0.00	0.0001
Present	108	67.92	193	100.00	
Typical morphology and distribution					
Absent	123	77.36	105	54.40	0.0001
Present	36	22.64	88	45.60	
Chronic or chronically relapsing dermatitis					
Absent	129	81.13	12	6.22	0.0001
Present	30	18.87	181	93.78	
Personal or family history of atopy					
Absent	112	70.44	12	6.22	0.0001
Present	47	29.56	181	93.78	
Minor Criteria					
Xerosis					
Absent	89	55.97	11	5.70	0.0001
Present	70	44.03	182	94.30	
Tendency toward cutaneous infections, impaired cell-mediated immunity					
Absent	152	95.60	166	86.01	0.002
Present	7	4.40	27	13.99	
Tendency toward hand or foot dermatitis					
Present	159	100.00	193	100.00	
Ichtyosis/Palmar hyperlinearity, keratosis pilaris					
Absent	151	94.96	110	56.99	0.0001
Present	8	5.03	83	43.01	
Immediate skin test reaction					
Absent	157	98.74	191	98.96	0.845
Present	2	1.26	2	1.04	
Elevated serum IgE					
Absent	158	99.37	188	97.41	0.157
Present	1	0.63	5	2.59	
Pityriasis alba					
Absent	150	94.34	108	55.96	0.0001
Present	9	5.66	85	44.04	
Nipple eczema					
Absent	152	95.60	172	89.12	0.025
Present	7	4.40	21	10.88	
Early-age of onset					
Absent	157	98.74	161	83.42	0.0001
Present	2	1.26	32	16.58	
Cheilitis					
Absent	157	98.74	177	91.71	0.003
Present	2	1.26	16	8.29	
Dennie-Morgan infraorbital folds					
Absent	151	94.97	168	87.05	0.011
Present	8	5.03	25	12.95	

Table 3. CONT.

Atopic dermatitis criteria of Hanifin&Rajka	OACD patients without atopic dermatitis (-)		OACD patients with atopic dermatitis (+)		p
	n=159	%	n=193	%	
Periorbital darkening					
Absent	145	91.19	125	64.77	0.0001
Present	14	8.81	68	35.23	
Facial pallor, erythema					
Absent	153	96.23	139	72.02	0.0001
Present	6	3.77	54	27.98	
Keratoconus					
Absent	159	100.00	193	100.00	
Recurrent conjunctivitis					
Absent	158	99.37	190	98.45	0.415
Present	1	0.63	3	1.55	
Anterior subcapsular cataract					
Absent	159	100.00	193	100.00	
White dermographism/delayed blanch					
Absent	157	98.74	161	83.42	0.0001
Present	2	1.26	32	16.58	
Perifollicular accentuation					
Absent	151	94.97	125	64.77	0.0001
Present	8	5.03	68	35.23	
Enviromental/emotional triggering					
Absent	124	77.99	91	47.15	0.0001
Present	35	22.01	102	52.85	
Intolerance to wool and lipid solvents					
Absent	142	89.31	97	50.26	0.0001
Present	17	10.69	96	49.74	
Food intolerance					
Absent	159	100.00	191	98.96	0.198
Present	0	0.00	2	1.04	
Itch when sweating					
Absent	155	97.48	174	90.16	0.006
Present	4	2.52	19	9.84	
Anterior neck lines					
Absent	154	96.86	170	88.08	0.002
Present	5	3.14	23	11.92	

*AD: Atopic dermatitis; **OACD: Occupational allergic contact dermatitis.

were female. No statistically significant differences were detected on the basis of gender, and the patients in both groups were in their thirties.

When the mean latency period is considered, the time from employment to first symptoms has been reported to be shorter in atopic cases than nonatopic OACD patients (71 vs 84 months).[2] Although we could not find any comparative study of OACD with and without AD, the period of 9-12 and >12 months from employment to first symptoms of OACD was significantly lower in atopic patients than nonatopics, with an increasing rate over time. This might suggest that coexistence of AD and OACD may prevent long-term employment of textile workers. Additionally, the presence of a statistically longer disease duration in the OACD group with AD suggested that these patients might acquire a contact sensitization sooner than others.

It is usually thought that white skin is more easily sensitized because a dense pigment network or thicker horny layer can lead to a decreased susceptibility to allergens.[20] We also found that there was significantly greater representa-

Table 4. Comparison of the responses for patch test allergens according to OACD* groups

Patch Test Allergens	OACD patients without atopic dermatitis (-)		OACD patients with atopic dermatitis (+)		p
	n=159	%	n=193	%	
Nickel Sulphate					
Absent	146	91.82	137	70.98	0.0001
Present	13	8.18	56	29.02	
Wool Alcohols					
Absent	156	98.11	174	90.16	0.002
Present	3	1.89	19	9.84	
Neomycin Sulphate					
Absent	155	97.48	183	94.82	0.203
Present	4	2.52	10	5.18	
Pottasium Dichromate					
Absent	151	94.97	174	90.16	0.091
Present	8	5.03	19	9.84	
Caine Mix					
Absent	154	96.86	183	94.82	0.346
Present	5	3.14	10	5.18	
Fragrance Mix					
Absent	154	96.86	172	89.12	0.006
Present	5	3.14	21	10.88	
Colophony					
Absent	159	100.00	183	94.82	0.004
Present	0	0.00	10	5.18	
Paraben Mix					
Absent	158	99.37	185	95.85	0.038
Present	1	0.63	8	4.15	
Negative Control					
Absent	158	99.37	192	99.48	0.891
Present	1	0.63	1	0.52	
Balsam of Peru					
Absent	153	96.23	179	92.75	0.160
Present	6	3.77	14	7.25	
Ethylenediamine dhydrochloride					
Absent	155	97.48	180	93.26	0.066
Present	4	2.52	13	6.74	
Cobalt Chloride					
Absent	145	91.19	170	88.08	0.343
Present	14	8.81	23	11.92	
13-p-tert-butylphenol formaldeyhde resin					
Absent	150	94.34	169	87.56	0.03
Present	9	5.66	24	12.44	
Epoxy Resin					
Absent	147	92.45	173	89.64	0.360
Present	12	7.55	20	10.36	
Carba Mix					
Absent	151	94.97	174	90.16	0.091
Present	8	5.03	19	9.84	

Table 4. CONT.

Patch Test Allergens	OACD patients without atopic dermatitis (-)		OACD patients with atopic dermatitis (+)		p
	n=159	%	n=193	%	
Black Rubber Mix					
Absent	153	96.23	178	92.23	0.115
Present	6	3.77	15	7.77	
Cl+Me+Isothiazolinone					
Absent	155	97.48	174	90.16	0.006
Present	4	2.52	19	9.84	
Quarternium-15					
Absent	155	97.48	180	93.26	0.066
Present	4	2.52	13	6.74	
Methyldipromo glutaronitrile					
Absent	156	98.11	186	96.37	0.328
Present	3	1.89	7	3.63	
20-p-Phenylene diamine					
Absent	146	91.82	175	90.67	0.705
Present	13	8.18	18	9.33	
Formaldehyde					
Absent	159	100.00	186	96.37	0.015
Present	0	0.00	7	3.63	
Mercapto Mix					
Absent	158	99.37	184	5.34	0.023
Present	1	0.63	9	4.66	
Thiomersal					
Absent	156	98.11	172	89.12	0.001
Present	3	1.89	21	10.88	
Thiuram mix					
Absent	155	97.48	182	94.30	0.141
Present	4	2.52	11	5.70	
Diazolidinyl urea					
Absent	156	98.11	182	94.30	0.069
Present	3	1.89	11	5.70	
Quinoline Mix					
Absent	155	97.48	186	96.37	0.551
Present	4	2.52	7	3.63	
Tixocortol-21-pivate					
Absent	157	98.74	185	95.85	0.105
Present	2	1.26	8	4.15	
Gold sodium thiosufate					
Absent	146	91.82	172	89.12	0.393
Present	13	8.18	21	10.88	
Imidazolidinyl urea					
Absent	156	98.11	184	95.34	0.153
Present	3	1.89	9	4.66	
Budesonide					
Absent	158	99.37	178	92.23	0.001
Present	1	0.63	15	7.77	
Hydrocortisone-17-butyrate					
Absent	159	100.00	188	97.41	0.041
Present	0	0.00	5	2.59	

Table 4. CONT.

Patch Test Allergens	OACD patients without atopic dermatitis (-)		OACD patients with atopic dermatitis (+)		p
	n=159	%	n=193	%	
Mercapto-benzothizaole					
Absent	158	99.37	190	98.45	0.415
Present	1	0.63	3	1.55	
Bacitracin					
Absent	154	96.86	190	98.45	0.319
Present	5	3.14	3	1.55	
Parthenolide					
Absent	159	100.00	188	97.41	0.041
Present	0	0.00	5	2.59	
Dispers Blue 106					
Absent	138	86.79	93	48.19	0.0001
Present	21	13.21	100	51.81	
2-Bromo-2-Nitropropane-1,3-diol					
Absent	154	96.86	180	93.26	0.128
Present	5	3.14	13	6.74	

*OACD: Occupational allergic contact dermatitis.

tion of the skin phototypes I and II in the OACD group with AD. Therefore, there is the possibility that the facilitating effect of a lighter skin type and the presence of AD may make these patients more susceptible to contact allergens.

When the subgroup distributions are examined in literature regarding textile OACD, Mathur et al.[19] reported that their patients were in dyeing, rinsing, and washing subgroups. Singhi et al.[18] indicated that most of their patients were in the dyeing sector,[18] whereas Chen et al.[17] reported on a group that was mostly comprised of sewing/ironing workers. The dyeing, accessory, and knitting subsectors were the most frequent subsectors seen among the participants of this study and they represented a significantly larger portion of the group with AD in comparison to the other group. Allergens present in these subsectors might have more bidirectional sensitization effects (both Type-1 and Type-2 reactions), than other sectors. However, existing studies on textile OCD are not comparable due to differences in the description of work sectors, screening periods, healthy-worker survivor effects, and because employees who experience occupational dermatitis are more likely to quit high-exposure jobs, either through termination of employment or changing their job at the same workplace.

Most ACD lesions are limited to the hands. Chan et al.[17] reported that the hands (82.4%) were the most frequent site affected by OCD in sewing and ironing workers.[17] In another study, the hands and wrists were determined to be the most affected areas in ironing workers.[21] The predominance of involvement was reported to be hands >flexor >extensor of forearm by Matura et al.[19] We obtained similar findings in both groups: The principle location in both groups was limited to the hands, and the difference was insignificant. Yet, our data also determined that the presence of lesions in the other 3 locations examined was significantly higher in the OACD group with AD. A theory of atopic predisposition might be supported by the greater involvement of the face, arms, and trunk locations in addition to the hands alone. Textile workers are frequently exposed many potentially harmful substances, such as metal tools, dyes, potassium dichromate, leather, aerosols, adhesives, and finishing agents.[8,17,21] OACD in textile workers is most often attributed to sensitization to textile/formaldehyde resins and disperse dyes/dye finishes, which are typically used to color mixed fabrics/fibers.[8,22]

Lisi et al.[23] stated that the prevalence of OACD caused by textile dyes ranged from 1.4% to 5.8%. Chen et al.[21] found that the most detected allergens in workers in the Chinese garment industry were nickel sulphate, cobalt dichloride, potassium dichromate, p-tert-butylphenol formaldehyde resin, and colophony. Azo and anthraquinone dyes have been reported to cause contact dermatitis in consumers. Matura et al.[19] most commonly detected red RC salt (5-chloro-0-anisidine hydrochloride) in the workers in their study. The most notable responses in our research were to nickel sulphate, cobalt chloride, 13-p-tert-butylphenol formaldehyde, and epoxy resins, whereas the least response was to the paraben

mix, colophony, methyldibromo glutaronitrile, and neomycin sulphate, in descending order.

ACD occurs as a result of a Type-IV reaction. In the sensitization phase, after the capture of an allergen by antigen-presenting cells, an activation of naive T cells and subsequent differentiation of antigen-specific memory T cells occurs in the regional lymphoid tissue.[13] They subsequently become active as a result of re-exposure to allergen/cross-reacting allergens, such as nickel, latex, or poison ivy, which is usually attributed to a T cytotoxic 1 (Tc1) or T helper 1 (Th1) response, though sometimes Th2, Th17, and Th22 responses may play a role in the pathogenesis.[13,22,24] OACD in the textile industry is usually ascribed to sensitization to dye/dye finishes and textile/formaldehyde resins.[8]

AD is a chronic, multifactorial disease caused by a combination of genetic, immune, and environmental factors, accompanied by barrier disruption. Contact sensitization is classically the result of a Th2-mediated, Type-I (immediate) inflammatory response caused by the aberrant production of IgE against normally nonpathogenic antigens. Exposure to the allergen may be through ingestion, injection, direct contact, or inhalation, and the reaction may be either local or systemic.[13,24,25]

Some studies have demonstrated the potential for shared immune pathways in AD and ACD, including Th1, Th2, Th9, Th17, and Th22 responses. Thus, it is currently thought that after the primary hypersensitization of the skin to an allergen as a result of a Type-I response, a second, delayed (Type-IV) inflammatory response may occur that develops through direct action of sensitized Th1 cells when stimulated by contact with antigens. An eczematoid reaction at the site of the contact is then triggered.[24,25] Moreover, some studies assessing the relationship between the 2 diseases have identified common allergens, including nickel, cobalt, potassium dichromate, chromium, lanolin, neomycin, formaldehyde, and fragrance markers in patients with both diseases, which may play a role in both Type-I and Type-IV sensitization as triggers.[24–26]

The results of the present study support the data indicating that AD and OACD can be seen together, and atopic diathesis may facilitate early development of OACD. However, our study is not without limitations. The relatively small number of participants, who were patients from a single clinic and employees from only 63 factories, may raise concerns regarding the validity of our findings. However, our hospital is located in a district that is one of the major manufacturing centers of the textile industry in our city, so we believe that our results can provide a general view on the topic.

Secondly, there was an unequal number of patients in the different subsectors because the study was conducted with randomly admitted patients. However, we believe that this inequality may be valuable in the sense of indicating the real distribution of OACD subjects, and particularly as an initial example of research conducted on this topic.

A third shortcoming was the inability to determine results for textile-specific allergens because they were unavailable. However, our results showed that, not only specific series, but also standard contact allergens, may play an important role in the development of textile-related OACD. Conducting more comprehensive studies on this subject with a large sample size can be difficult due to differences in exposed substances, subsectoral conditions, workplace rotation and short-term employment; however, there is a need for broad-based, controlled studies to better analyze the topic.

Conclusion

Given the increasingly varied types of materials used in textile manufacturing, a better understanding of individual or workplace-mediated predisposing factors for OACD is necessary in order to take targeted measures. The coexistence of AD and certain contact allergens may facilitate earlyonset OACD in textile employees, especially those working in certain subsectors. With awareness of the factors that lead to exacerbation in OACD, the required measures can be taken without delay. It would also be helpful to investigate workers' atopic predisposition during the recruitment phase in order to avoid loss of labor.

Disclosures

Ethics Committee Approval: The study was approved by the Local Ethics Committee.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Authorship Contributions: Concept – B.T.; Design –B.T.; Supervision –B.T., I.K.A.; Materials –B.T.; Data collection &/or processing – B.T., I.K.A.; Analysis and/or interpretation – B.T.; I.K.A.; Literature search – B.T.; Writing – B.T.; Critical review – B.T., I.K.A.

References

1. Odhiambo JA, Williams HC, Clayton TO, Robertson CF, Asher MI; ISAAC Phase Three Study Group. Global variations in prevalence of eczema symptoms in children from ISAAC Phase Three. J Allergy Clin Immunol 2009;124:1251–8. [CrossRef]
2. Spiewak R. Contact dermatitis in atopic individuals. Curr Opin Allergy Clin Immunol 2012;12:491–7. [CrossRef]
3. Herro EM, Matiz C, Sullivan K, Hamann C, Jacob SE. Frequency of contact allergens in pediatric patients with atopic dermatitis. J Clin Aesthet Dermatol 2011;4:39–41.

4. Kirchhof MG, de Gannes GC. Atopy Associated With Positive Patch Test and Possible Allergic Contact Dermatitis. J Cutan Med Surg 2018;22:405–10. [CrossRef]
5. Malajian D, Belsito DV. Cutaneous delayed-type hypersensitivity in patients with atopic dermatitis. J Am Acad Dermatol 2013;69:232–7. [CrossRef]
6. Tada J. Diagnostic Standard for Atopic Dermatitis. Japan Med Assoc J 2002;45:460–5.
7. Proksch E, Brasch J. Abnormal epidermal barrier in the pathogenesis of contact dermatitis. Clin Dermatol 2012;30:335–44. [CrossRef]
8. Wigger-Alberti W, Elsner P. Occupational Contact Dermatitis in the Textile Industry. In: Elsner P, Hatch K, Wigger-Alberti W, editors. Textiles and the Skin, Curr Probl Dermatol, 1st ed. Basel: Karger; 2003. p. 114–22. [CrossRef]
9. Heinzerling L, Mari A, Bergmann KC, Bresciani M, Burbach G, Darsow U, et al. The skin prick test - European standards. Clin Transl Allergy 2013;3:3. [CrossRef]
10. Tanno LK, Darlenski R, Sánchez-Garcia S, Bonini M, Vereda A, Kolkhir P, et al. International survey on skin patch test procedures, attitudes and interpretation. World Allergy Organ J 2016;9:8.
11. Nicholson PJ. Evidence-based guidelines: occupational contact dermatitis and urticaria. Occup Med (Lond) 2010;60:502–4. [CrossRef]
12. Thyssen JP, Johansen JD, Linneberg A, Menné T, Engkilde K. The association between contact sensitization and atopic disease by linkage of a clinical database and a nationwide patient registry. Allergy 2012;67:1157–64. [CrossRef]
13. Uehara M, Sawai T. A longitudinal study of contact sensitivity in patients with atopic dermatitis. Arch Dermatol 1989;125:366–8.
14. Mailhol C, Lauwers-Cances V, Rancé F, Paul C, Giordano-Labadie F. Prevalence and risk factors for allergic contact dermatitis to topical treatment in atopicdermatitis: a study in 641 children. Allergy 2009;64:801–6. [CrossRef]
15. Czarnobilska E, Obtulowicz K, Dyga W, Spiewak R. A half of schoolchildren with 'ISAAC eczema' are ill with allergic contact dermatitis. J Eur Acad Dermatol Venereol 2011;25:1104–7. [CrossRef]
16. Green CM, Holden CR, Gawkrodger DJ. Contact allergy to topical medicaments becomes more common with advancing age: an age-stratified study. Contact Dermatitis 2007;56:229–31. [CrossRef]
17. Chen YX, Cheng HY, Li LF. Prevalence and risk factors of contact dermatitis among clothing manufacturing employees in Beijing: A cross-sectional study. Medicine (Baltimore) 2017;96:e6356.
18. Singhi MK, Menghani PR, Gupta LK, Kachhawa D, Bansal M. Occupational contact dermatitis among the traditional 'tie and dye' cottage industry in Western Rajasthan. Indian J Dermatol Venereol Leprol 2005;71:329–32. [CrossRef]
19. Mathur NK, Mathur A, Banerjee K. Contact dermatitis in tie and dye industry workers. Contact Dermatitis 1985;12:38–41. [CrossRef]
20. Ale SI, Maibach HI. Operational definition of occupational allergic contact dermatitis. In: Kanerva L, Elsner P, Wahlner JE, Maibach HI, editors. Handbook of Occupational Dermatology. 1st ed. Heidelberg: Springer; 2000. p. 637–43. [CrossRef]
21. Chen YX, Gao BA, Cheng HY, Li LF. Survey of Occupational Allergic Contact Dermatitis and Patch Test among Clothing Employees in Beijing. Biomed Res Int 2017;2017:3102358. [CrossRef]
22. Dhingra N, Shemer A, Correa da Rosa J, Rozenblit M, Fuentes-Duculan J, Gittler JK, et al. Molecular profiling of contact dermatitis skin identifies allergen-dependent differences in immune response. J Allergy Clin Immunol 2014;134:362–72. [CrossRef]
23. Lisi P, Stingeni L, Cristaudo A, Foti C, Pigatto P, Gola M, et al. Clinical and epidemiological features of textile contact dermatitis: an Italian multicentre study. Contact Dermatitis 2014;70:344–50.
24. Werfel T, Allam JP, Biedermann T, Eyerich K, Gilles S, Guttman-Yassky E, et al. Cellular and molecular immunologic mechanisms in patients with atopic dermatitis. J Allergy Clin Immunol 2016;138:336–49. [CrossRef]
25. Simonsen AB, Johansen JD, Deleuran M, Mortz CG, Sommerlund M. Contact allergy in children with atopic dermatitis: a systematic review. Br J Dermatol 2017;177:395–405. [CrossRef]
26. Simonsen AB, Deleuran M, Johansen JD, Sommerlund M. Contact allergy and allergic contact dermatitis in children - a review of current data. Contact Dermatitis 2011;65:254–65.