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A DOUBLE CASE REPORT: CLINICALLY DIFFERENT OCCUPATIONAL DERMATOSES RESULTING FROM IDENTICAL EXPOSURE TO WORK ENVIRONMENT AT A PHOTOGRAPHIC LABORATORY

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Abstract: This paper presents the case of two workers employed in a photographic laboratory. Despite that the degree of occupational exposure to photographic chemicals, as well as gender and age were the same in both patients, they developed dermatoses different in morphology and clinical course. This observation suggests that individual susceptibility plays a very important role in etiopathogenesis of dermatoses caused by photographic chemicals.

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INTRODUCTION

Photographic chemicals, especially those used in colour photography processing, are well known, potent sensitizing agents [17, 21, 23, 24, 25]. The most common adverse reactions resulting from recurrent exposure to photochemicals are allergic contact dermatitis [3, 5, 6, 9, 12, 14, 19] and lichen planus-like eruptions [1, 2, 13, 18, 20, 26]. Contact urticaria from photochemicals was also described [11]. The difference between contact dermatitis and the lichenoid eruptions is not clear. The clinical symptoms of so-called photographer's lichen are similar to those of the drug-related lichen planus [18] and some investigators reported the histology as compatible with lichen planus [1, 2]. Other authors, however, stress that the histological picture of the affected skin resembles changes occurring in contact dermatitis rather than in typical lichen planus [10, 20].

CASE REPORT I

P. A., female, born 1975. For two years the patient worked in a photographic laboratory at the manual and automated processing of black-white and colour films, and film printing. During the work she had continuous contact with following substances: 2-naphtylamine, amidole, mercury chloride, diazolethyloaniline, potassium dichromate, fenidone, formaldehyde, hydroquinone, metol, sodium methylodisulfate, hydroxylamine sulfate and mercury salts. In September 1993, two months after the introduction of new processing technology based on Tetenal® photochemicals, the first skin lesions appeared. On the right wrist, itching and reddish macula were noted, subsequently with furfuraceous desquamation and exudating papules. The changes gradually extended over the upper half of the body, involving the upper extremities, chest and abdomen. Erythema and oedema

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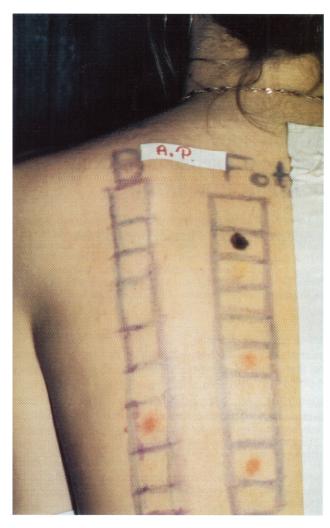


Figure 1. Patient P. A. Patch test results after 48 hours. Positive reactions are seen to paraaminoazobenzene (laterally) and to mercury chloride and IPPD (in medial series).



Figure 2. Patient P. A. Open application test on the left forearm, after 24 hours. Positive reactions are seen to NC ORWO colour developer, NC ORWO replenisher, and Tetenal CD-S.

Table 1. Results of patch testing with photographic chemicals from the patients' workplace. Chemicals marked with asterisks are commercial preparations whose composition is not known being covered by patents. Abbreviations: pet = petrolate, aq = distilled water, IPPD = N-isopropyl-N'-phenyl-paraphenylenediamine.

Chemicals tested	Concentration	P. A.		G. Ż.	
	(solvent)	48 h	72 h	48 h	72 h
Hydroquinone	0.2% (pet)	-	-	+	++
Hexamethylenetetraamine	2% (pet)	_	-	-	-
Aniline	1% (pet)	_	-	-	-
Metol	1% (pet)	_	-	+	+
Hydrazine	1% (pet)	_	_	-	-
Benzidine	1% (pet)	_	_	_	_
Paraaminoazobenzene	1% (pet)	+	++	_	_
Sodium sulfite	1% (pet)	_	-	-	-
Phenylhydrazine	0.2% (pet)	_	_	-	-
Nickel sulfate	5% (aq)	_	_	-	-
Paraphenylenediamine	1% (pet)	_	_	_	_
Potassium dichromate	0.5% (aq)	_	_	-	-
Formaldehyde	0.7% (aq)	_	-	-	-
Mercaptobenzothiazole	0.03% (aq)	_	-	-	-
Mercury chloride	2% (pet)	+	++	++	++
Resorcinol	2% (pet)	_	_	-	-
Sulfathiazole	5% (pet)	_	_	-	-
IPPD	0.1% (pet)	+	++	++	++
Ethylenediamine	10% (pet)	_	_	-	-
C-41 Tetenal colour developer*	1% (aq)	_	-	_	-
CD-R Tetenal colour replenisher*	1% (aq)	+	+	_	-
BX Tetenal photographic reducer*	1% (aq)	-	-	-	-
C-41 Tetenal fixing solution*	1% (aq)	_	_	_	-
Foton black-white paper developer*	1% (aq)	+	+	-	-
Foton black-white film developer*	1% (aq)	_	-	-	-
Foton black-white fixing solution*	1% (aq)	+	+	-	-

gradually appeared on the face and neck. Because the skin changes tended to be exacerbated, the patient was hospitalized in November 1993 in the dermatological ward, where allergic and seborrhoeic dermatitis was diagnosed. After leaving hospital, the patient was admitted to the Allergy Outpatient Clinic.

Physical examination on the first visit: Erythemosquamous lesions with exudating papules on the skin of the upper half of the body, except the back. No other abnormal findings.

The skin prick tests with common allergens (Biomed Poland, Allergopharma Germany) were done with the following results: solvent (wheal 3 mm/erythema 0 mm), histamin (5/20), *Dermatophagoides pteronyssinus* (4/25), *Dermatophagoides farinae* (3/20), house dust (3/25).

Table 2. Results of open application tests with commercial preparations of photographic chemicals used in the laboratory. Composition of these chemicals is not known being covered by patents. Abbreviations: aq = distilled water.

Chemicals tested	Concentration	P. A.		G. Ż.	
	(solvent)	24 h	48 h	24 h	48 h
NC ORWO colour developer	1% (aq)	-	-	-	-
NC ORWO fixing solution	1% (aq)	+	?	_	_
NC ORWO replenisher	1% (aq)	+	?	+	+
NC ORWO reducer	1% (aq)	_	-	-	-
C-41 reducer	1% (aq)	_	-	-	-
Tetenal CD-S	1% (aq)	+	+	++	++
Formaldehyde	1% (aq)	+	_	_	_
Tetenal C-41 BL-S	1% (aq)	-	_	+	++

Response to other allergens (pollens, dusts, flour, meat, fish, vegetables) was negative. Standardized photographic chemicals, metal compounds and organic dyes for patch testing were obtained from the E. Jaworski firm, Katowice, Poland. Other chemicals tested were commercial preparations obtained from the photographic laboratory and prepared by diluting with distilled water to 1%. The chemicals were applied to patches of filter paper measuring 1×1 cm. The patches were then placed onto the skin and attached using hypoallergenic adhesive tapes. After 48 hours the tapes were removed and the skin reaction was observed (Fig. 1). The next check followed 24 hours later. In order to control chemicals suspected of having an irritating effect on the skin, we used the open application test instead of patch tests. The substance tested was placed on the skin of the forearms, and allowed to dry. The effect was observed after 24 and 48 hours (Fig. 2). The results were recorded using the following interpretation scheme: -= negative reaction, ? = doubtful reaction (light redness on the contact area), += weak reaction (erythematous only), ++ = strong (oedematous or vesicular) reaction, +++ = extreme (bullous or ulcerative) reaction. The results of skin testing with the chemicals are shown on Table 1 (patch tests) and Table 2 (open application tests). The cessation of work at the photographic laboratory resulted in complete clearance of skin lesions within two months.

Diagnosis. On the basis of the patient's history, clinical picture and results of provocative skin tests with chemicals from the work environment, we have diagnosed allergic contact dermatitis due to sensitization to multiple photochemicals.

CASE REPORT II

G. Ż., female, born 1977. For one year the patient worked in the same photographic laboratory with the same responsibilities as the patient described above. Occupational exposure was therefore identical. In September 1993, at nearly the same time as in patient P. A., reddish, dry macules appeared on the hands. After contact with water, burning clusters of exudating papules



Figure 3. Patient G. Ż. Positive patch test reactions are seen with hydroquinone, metol, mercury chloride and IPPD.

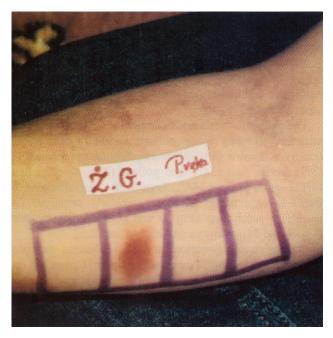


Figure 4. Patient G. Ż. Open application test on the right forearm, after 24 hours. Visible reaction to NC ORWO replenisher.

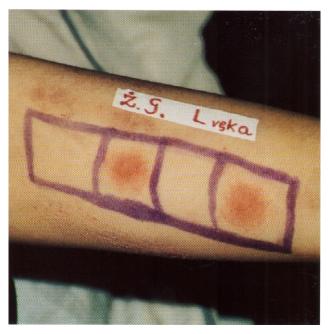


Figure 5. Patient G. Ż. Open application test on the left forearm, after 24 hours. Positive reactions to Tetenal CD-S and Tetenal C-41 BL-S. Note skin lesions surrounding test area on Figures 4 and 5. The photographs were taken three months after cessation of work and show lesions typical for patient G. Ż.

developed on the skin lesions. These changes primarily involved the palmar region only, subsequently spreading to the arms and chest. These particular lesions persisted for over a month, resolving into xerous, reddish-brown hyperpigmented macules which gradually turned pale. Increased susceptibility persisted in these regions which manifested itself as a strong burning sensation on contact with water or after mechanical injury. Each visit to her workplace resulted in a significant worsening of the skin status, and the appearance of new foci of lesions. In October 1993, she was hospitalized twice in the dermatological ward. Eczema and pyodermia were diagnosed, and a certificate of work disability was given.

Physical examination during the first visit: On the upper half of the body, except the back, erythemo-squamous reddish-brown lesions with tiny papules. No other abnormal findings.

The skin prick tests with common allergens were done with the following results: solvent (wheal 0 mm/erythema 0 mm), histamine (6/0), *Dermatophagoides pteronyssinus* (3/0), house dust (3/0), tree pollens (3/0), weed pollens (4/12), grass pollens (4/0). Tests with other allergens, as listed in case report I, were completely negative. The results of patch tests (Fig. 3) and open application tests (Fig. 4 and 5) are shown on Tables 1 and 2.

Despite work cessation, the skin changes persisted during the entire follow-up period (nearly 10 months), and susceptibility to new factors, such as drugs, cosmetics, physical factors, developed. The morphology of lesions gradually changed, turning into large, brownish, vascular macules with slight atrophy. The patient, however, refused the taking of skin sample for histological examination.

Diagnosis. The patient's history, clinical picture and results of the skin tests with photochemicals led us primarily to diagnose lichenoid contact dermatitis provoked by multiple photochemicals.

DISCUSSION

At the introduction of new, automated technologies a decrease in the occurrence of occupational dermatoses among workers in photographic laboratories was expected [15, 16, 24]. Paradoxically, two months modernization in the laboratory described, both employees developed severe dermatoses. The coincidence with modernization is striking. In this particular case, however, the new automated processing unit was placed in the laboratory where manual processing continued. This probably resulted in an increase in the concentration of chemical vapours. Moreover, the technical supervisor stated that in this room the exhaust system was inefficient, and both women worked uncautiously without protecting gloves. It should be stressed here that in both cases the first skin lesions appeared on the hands. In both women, the components of photochemicals used for a long time in this laboratory (Tab. 1) provoked reactions comparable to the new chemicals.

There is controversy about the etiopathogenesis of dermatoses from colour developers. Some investigators believe that they may be due to direct contact with the chemicals [10], while others believe that systemic absorption is responsible for the lesions [2, 7]. The distribution of lesions raises the question of whether the agents can produce skin eruption by absorption of fumes or by direct contact. In our patients, the skin changes were restricted to skin areas exposed to vapours and did not occurred on the legs or back. This suggests a possibility of the induction of skin changes by vapours. It should be stressed, however, that the first lesions appeared on skin areas exposed to direct contact with chemicals. We surmise therefore that direct contact may have a priming effect for evoking changes which subsequently develop following the exposure to vapours. Referring to the past history and to present disorders, both of the presented patients show a remarkable similarity, but interestingly there was a difference in the clinical picture of their dermatoses. Both patients, working together, were exposed to the same chemicals with identical frequency and duration. In the patients, the coincidence of the onset of the disease and of the introduction of new processing technology is also remarkable. During the two months after modernization, both women developed severe dermatoses for the first time in their lives.

Despite the duration and intensity of exposure and onset time of symptoms were almost identical, the course of the diseases differed significantly. This seems to negate the suppositions that the clinical picture of skin disorders caused by photographic chemicals depends mainly on the duration of exposure [22], or type of chemical responsible [25]. The individual's age also could not play a role, as

there was only a two year difference between the observed patients. Moreover, the patients revealed hypersensitivity to different chemicals, which again indicates a crucial role of the individual predisposition, as the external conditions were identical. Both women showed symptoms of mild atopy, but based on her past history, patient P. A. revealed more signs of atopic diathesis. This difference may be of importance, because a relationship between atopic predisposition and disturbances of cellular immunity was found [8].

The fact that the patients were sensitive to different, not the same, photochemicals used in their laboratory is an argument against toxic etiology of skin lesions. Moreover, the substances tested were diluted far more than met in working conditions. The *in vitro* tests with patients' lymphocytes were not necessary, as the diagnosis could be established with patch tests at much lower costs [4]. We surmise that skin biopsies would be helpful but both patients refused the taking of skin samples.

Some aspects of the case of patient G. -. need discussion. Typically, despite the fact that the photochemicals-induced lichenoid lesions have the clinical appearance of lichen planus, they are far more amenable to therapy than the idiopathic form of the disease [18, 26] and complete clearance of the rashes follows within 1-3 months after avoiding exposure [7, 10]. It has also been reported that the lichenoid changes may persist "six months or a year"; however, in this report [2] it was not stated if the patients described continued their work with photochemicals or not. A weak response to the standard therapy, the persistence of skin lesions despite cessation of work, and an increasing number of precipitating factors not related to the work environment, argued against typical lichenoid contact dermatitis in patient G. -. We surmise that it may be an autoimmune skin disease provoked by photochemicals. The patient, however, discouraged by unsatisfactory therapeutic results, refused further diagnostic procedures.

CONCLUSION

The conclusion of this paper is that aside irritating and allergenic properties of photochemicals, individual susceptibility also plays an important role in etiopathogenesis and the determination of a clinical picture of dermatoses caused by these chemicals.

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