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A Case of Extreme and General Cutaneous Light Sensitivity in Combination with so-called 'Screen Dermatitis' and 'Electro-sensitivity' - a Successful Rehabilitation after Vitamin A Treatment - a case report

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Abstract

An increasing number of patients has recently been described as suffering from subjective and objective skin- and mucosa-related symptoms after exposure to electromagnetic devices, such as visual display terminals and mobile telephones. Our objective in this study is to report on one such patient having in addition a profound and general light-sensitivity, and to provide a cellular basis for further clinical judgement. Immunohistochemistry using different cellular markets was employed. It was found that protein S-100 and HLA-DR-positive dendritic cells were dramatically decreased in number, especially in the epidermis. From our preliminary data, it seems likely that certain paramount cellular changes may be present in this patient category claiming to suffer from electromagnetic field exposure. The exact cause as well as the exact cellular and molecular basis of this disorder, however, is at present not understood. Key words: dermatology; immunohistochemistry; dendritic cells; peptides; light sensitivity; screen dermatitis; electrosensitivity.

Abbreviations: CGRP, calcitonin gene-related peptide; EMFs, electromagnetic fields; 5-HT, serotonin; HLA-DR, human histocompatibility complex class II (subregion DR); NGFr, nerve growth factor receptor; NSE, neuron-specific enolase; PGP 9.5, protein gene product 9.5; S-100, protein S-100; TRITC, tetramethylrhodamine-isothiocynate isomer R; VDTs, visual display terminals; VIP, vasoactive intestinal polypeptide.

Introduction

In the past decade an increasing number of patients who claim to suffer from different adverse health symptoms, after exposure to visual display terminals (VDTs), mobile telephones, high tension power lines as well as other electromagnetic devices, both at their work and in their home, has been described in the literature[1,2]. These include both subjective and objective skin- and mucosa-related symptoms, such as itching, smarting, pain, heat sensation, redness, papules, pustules, etc. Some patients also have symptoms from internal organ systems, such as the heart and the central nervous system. We use the term 'screen dermatitis' to describe these patients, although any clear-cut evidence for a casual relation is still lacking. Clinical dermatologists often describe these patients as suffering from either some kind of previously acknowledged skin disease, e.g. seborrhoeic dermatitis or rosacea, or from so-called 'techno-stress', a term first used in Japan for work-related stress. Also Pavlovian-type conditioning has been attributed to this group of patients[3]. Since very little is known about the exact cause of the above-mentioned symptoms, unfortunately, generally very little treatment can be offered.

Case Report

A 47-year-old woman claiming to suffer from 'screen dermatitis' presented in June 1994 an extreme general light sensitivity. She had objective and subjective skin, heart and headache symptoms when exposed to VDTs as well as presumably to electromagnetic fields (EMFs) from internal house wiring.

She first developed her acute 'screen dermatitis' symptoms (itch, skin burning sensation, flare, headache, fatigue, etc.) in May 1989, after having had single outbursts during the autumn of 1988 as well as during early spring 1989. The patient herself suggested the removal of several amalgam fillings and parallel sunbed use to be the cause of the sudden outburst of the severe 'screen dermatitis' in May 1989.

She became slightly better during the spring of 1990, but returned to the handicapped situation in June 1990. Thereafter, the patient has had positive and negative phases in her disease history; however, the general trend has been negative.

At the end of June 1994, after a short (½ hour) exposure to sunlight on her back through a glass window, the patient suddenly developed a severe, general and rapidly increasing sensitivity to all light. She complained of a strongly burning sensation in her skin (later also in "other, more deeply located, tissues"). However, upon inspection, the skin looked normal and often felt normal in temperature. The patient started to protect herself with dark, thick clothing, was forced to stay indoors in complete or near-complete darkness and only left her home for short walks in shady forests during late evening when the sun had set and there is (in Sweden) more or less complete darkness. At first, some areas of the body felt more involved than others (the hands, feet, thighs and face). The patient did not have an elevated body temperature. It may be noted, that in connection with the onset of the 'screen dermatitis' in 1989, the patient, having been sensitive to cold, suddenly started to tolerate lower indoor temperatures, even as low as 14° - 17° C. Also, she became very sensitive to light and lived in darkness for a couple of months. This time, the patient recovered by treatment with a concentrate of β-carotene. β-carotene had no effect in 1994. An interesting detail is that she did not report her eyes to be more sensitive to normal levels of light, but if her eyes were exposed to very strong light, it caused a dramatic increase in the total body light sensitivity. No visible, primary or secondary, skin lesions could be found upon physical examination.

Beginning in January 1995, the patient was advised to take a large dose of vitamin A (50,000 IU/day; initially every day for 3 months, then continuing during 14-day interval periods (14 days on, 14 days off)) combined with physical therapy, including massage, acupuncture as well as physical exercise. During this period, she gradually rehabilitated. In the Summer of 1996, she could expose herself to indirect sunlight (i.e., standing in the shade or walking around in the late afternoon), and in May 1998 she was feeling much better. The previous light exposure-related symptoms had more or less disappeared; however, she could still not tolerate standing in front of the electric oven, using the vacuum cleaner, talking on the telephone for any significant length of time, watching TV, etc. It may be noted that the use of selenium for a short time period at the early onset of the 'screen dermatitis', turned out to have a reverse effect on the symptoms, although later it had no effect. Clomipramine (Anafranilâ; 10-30 mg/day) combined with flupenthixol (Fluanxolâ; 0.25 mg/day) during 9 months of 1993 had no effect.

14

13

In December 1994 (during the worst period) and again in June 1996 (after the rehabilitation had occurred), the patient was subjected to two skin punch biopsies (3 mm), under local anaesthesia (mepivacaine; 5 mg/ml), from the right thigh (just above the knee). For comparative purposes, corresponding biopsies were also taken from normal healthy (matched) volunteers. The specimens were immersed in formalin/picric acid (for all markers, except histamine), or in carbodiimide (for histamine), diluted in 0.1 M Sörensen's phosphate buffer (pH 7.4) at 4° C, and then rinsed in the same buffer containing 10% sucrose for at least 24 hours. Perpendicular, 14 mcm cryostat sections were processed for indirect immunofluorescence[4]. The characteristics of the primary antibodies and their dilutions are stated in Table I. The sections were incubated overnight at 4° C in a humid atmosphere, followed by an incubation for 30 min. at 37° C with tetramethylrhodamine-isothiocyanate isomer R (TRITC) - conjugated donkey anti-rabbit or donkey anti-mouse IgG antiserum diluted 1.80. Control sections were incubated with the secondary antisera only or with primary antibodies absorbed by the antigen. The stained sections were blind-coded and independently evaluated by two investigators.

To our great surprise, protein S-100 and HLA-DR-positive dendritic cells were dramatically decreased in number in the first biopsy (from the worst period), especially in the epidermis (Fig. 1B, E). Also, their fluorescence intensity was decreased compared to healthy control material (Fig 1A, D).

The processes of the dendritic cells were fewer in number and thinner. However, the cellular morphology and number of the later (after rehabilitation) biopsy appeared similar to normal (Fig. 1C, F). The other markers (CGRP, NGFr, NSW, PGP 9.5, 5-HT, VIP) did not reveal any significant differences, including the mast cell marker histamine.

Discussion

From these studies, it is clear that the number of dendritic cells, especially of the epidermis, was decreased in the patient. This may indicate that these cells could be involved in the pathogenesis of the light-related symptoms. The only conditions revealing a similar cellular picture, which spring to our mind, are ultraviolet-, grenz ray- or conventional X-ray-induced damage to the cutaneous immune system as, for instance, monitored in the epidermal Langerhans cell population[5,6]. What is even more interesting is, of course, the potential carcinogenesis brought about by such irradiations[7,8]. Also, our own previous observations[9] from an open-field situation are of great importance, where the effect of EMFs from an ordinary RV set (duration: 30, 60 or 210 minutes; distance 50 cm) on the cellular/ neuronal populations of the skin of sampled patients (n=2) was investigated. In the biopsies taken before provocation, a remarkably high number of somatostatin immuno-reactive dendritic cells were found in the dermis, preferentially around the blood vessels and hair follicles as well as in the basal layer of the epidermis. After provocation, no somatostatin immunoreactive cells at all could be found in the patients investigated, using the presently employed immunohistochemical method.

Vitamin A has been demonstrated to be more effective than anything else for this patient. During the vitamin A treatment, the patient was to a large extent rehabilitated regarding her general light sensitivity; however, she was still sensitive to the presence of electric equipment, although not as much as before. The metabolism of vitamin A should be considered, since, in the human visual system, vitamin A is converted to α-cis-retinol, which is an essential chromophore component of rhodopsin, the photoreceptor protein of the retinal rods and is therefore essential for human vision. Maybe vitamin A influences cutaneous (as well as other) cellular systems similar to the retina. One explanation is that the patient for a time lost her melanocytes (or melanocytic content), as seen with the S-100 immunofluorescence, in response to an external or internal provocation. As a reaction to this, also her HLA-DR positive dendritic cells were affected. The vitamin A may have been capable of restoring this balance, at least partially.

The cellular and molecular basis for 'screen dermatitis' is not, at present, understood, and therefore neither is the cause. Whether this is due to electric or magnetic fields, a surrounding airborne chemical, stress factors, Pavlovian mechanisms, or something else, still remains an open question. However, it is evident from our preliminary data, that certain profound cellular changes may be present in the 'screen dermatitis' patients suffering from EMF exposure, including visible light.

Acknowledgements

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16

Fig.1. Legend: Immunofluorescence micrographs of normal (A, D) skin from a healthy volunteer, as well as early (= worst period) (B, E) and late (= after rehabilitation) (C, F) biopsies of the patient's skin after inclubation with protein S-100 positive epidermal dendritic cells, as well as in E, the strong reduction in the corresponding epidermal HLA-DR immunoreactive dendritic cells. In the late stage (C, F), the picture is similar to normal. e = epidermis. Magnification in A = D = E, and B = C = F. Bars in A and B indicate 50 mcm.

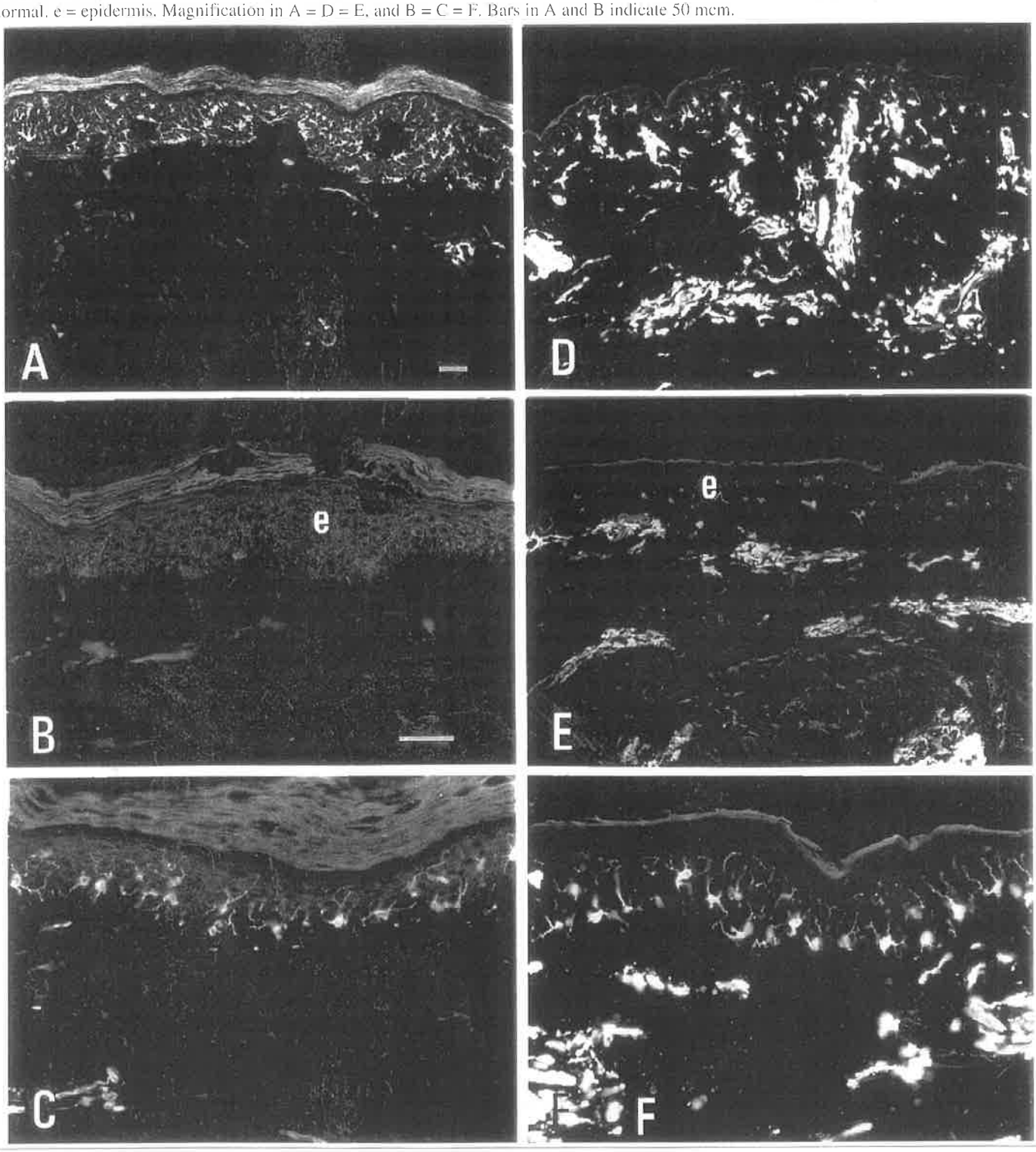

Table I. Description of the primary antibodies

Antibody	Abbreviation	Dilution	Species	Manufacturer
Calcitonin Gene-related peptide	CGRP	1:600	rabbit	Peninsula
Histamine	-	1:2,000	rabbit	Milab
Human histocompatibility complex class II (subregion DR)	HLA-DR	1:200	mouse	Dakopatts
Nerve growth factor receptor	NGFr	1:200	mouse	Amersham
Neuron-specific enolase	NSE	1:1,200	rabbit	UC
Protein gene product 9.5	PGP 9.5	1:2,000	rabbit	UC
Protein S-100	S-100	1:1,600	rabbit	K. Haglid, Göteborg & L. Olson, Stockholm
Serotonin	5-HT	1:600	rabbit	A Verhofstad, Nijmegen
Vasoactive intestinal polypeptide	VIP	1:1,600	rabbit	Peninsula

E R R A T A - Please, note the following typographical errors:

1) The title is wrong: Should read "A Case of Extreme and General Cutaneous Light Sensitivity in Combination with so-called "Screen Dermatitis" and "Electrosensitivity" - Successful Rehabilitation after Vitamin A Treatment"

2) The affiliation for Lennart Wetterberg should be changed from "...St. Göran´s...", to "...S:t Göran's..."

3) Under "Abstract", page 13, line 8, change "...markets..." into "...markers...", and "...protein S-100..." into "...protein S-100-..."

4) Under "Abbreviations", page 13, line 6, change "...-isothiocynate..." into "...-isothiocyanate..."

5) Under "Case Report", page 13, 2nd and 3rd line from bottom of the right column, change "....Anafranila..." into "...Anafranil®..." and "...Fluanxola..." into "...Fluanxol®..."

6) Under "Case Report", page 14, line 10 of the left column, change "...14 mcm..." into "...14 µm..."

7) Under "Case Report", page 14, line 17 of the left column, change "...1.80." into "...1:80."

8) Under "Case Report", page 14, line 21 of the left column, change "...protein S-100..." into "...protein S-100-..."

9) Under "Case Report", page 14, line 25 of the left column, continue the text directly after "...(Fig. 1A, D)." with the sentence "The processes of the dendritic cells were fewer..."

10) Under "Case Report", page 14, line 29 of the left column, change "...NSW,..." into "...NSE,..."

11) Under "Discussion", page 14, line 11 of the left column, change "...RV..." into "...TV..."

12) Under "Discussion", page 14, line 15 of the left column, change "...immunoreactive dendritic cells were found..." to "...immunoreactive dendritic cells was found..."

13) Under "Discussion", page 14, line 27 of the left column, change "...α-cis-retinol..." into"...11-cis-retinal..."

14) Under "Acknowledgements", page 14, line 3, change "...Industritjänstemannoförbundet..." to "...Industritjänstemannaförbundet..."

15) Under "References", page 14, ref. no. 4, line 3, change "...Histo Chem J..." into "...Histochem J..."

16) In Ref. no. 5, change "...(Stckh)..." to "...(Stockh)..."

17) Under "Fig. 1. Legend:", page 16, line 2, change "...after inclubation with protein S-100 positive..." into "...after incubation with protein S-100 (A-C) or HLA-DR (D-F) antiserum. Note in B the complete absence of S-100 positive..."

18) Under "Fig. 1. Legend:", page 16, line 4, change "...50 mcm." into "...50 µm."

19) In the Table, change "Calcitonin Gene-related..." into "Calcitonin gene-related..."