CHLORACNE AND ITS CLINICAL AND LABORATORY IMPLICATIONS IN TWO PATIENTS WITH 2,3,7,8-TCDD INTOXICATION IN VIENNA

Alexandra Geusau, Michael Meixner, Steffen Sandermann, Jolanta B Schmidt*, Erwin Tschachler and Georg Stingl

Department of Dermatology, Division of Immunology, Allergy and Infectious Diseases (DIAID), * Division of Special and Environmental Dermatology, University of Vienna Medical School, Währinger Gürtel 18-20, 1090 Vienna, Austria.

Introduction

A number of polymorphous signs and symptoms have been attributed to acute intoxication with 2,3,7,8-TCDD, but in man the most severely, and in most cases, exceptionally involved organ, is the skin (*1*). The distribution of chloracne lesions is of diagnostic importance (*2*). Its severity is reported to correlate well with the estimated cumulative TCDD concentrations after exposure (*3*). A change in differentiation of sebocytes as well as of keratinocytes of the follicular openings leading to follicular hyperkeratosis is responsible for the pathogenesis of chloracne and the consecutive cyst formation (*4,5*).Here we report on the clinical and laboratory course in two patients with 2,3,7,8-TCDD induced chloracne who were diagnosed at our department in April 1998. Shortly after they had moved into a newly adapted office in a textile research institute in December 1997 the two young women developed acne lesions in the face and gastrointestinal symptoms like nausea, vomiting and epigastric pain, which consequently had led to weight loss. The acute worsening of the more severely affected patient's condition led to her admittance to our ward in March 1998.

Materials and Methods

Over an observation period of approximately one year the two patients were monitored clinically and by laboratory and apparative investigations. A skin biopsy was taken from the pre-auricular area in the first patient.

Results and Discussion

When the first patient presented with a painful inflammation and edema of the centrofacial area, fever, myalgia and arthralgia in March 1998, the initial diagnosis of *acne fulminans* was established and treatment with high dose steroids and antibiotics intravenously was initiated. This led to a significant decrease of the acute inflammation, however, a progression of the follicular lesions was observed within the following 2-3 weeks: hundreds of cysts, open and closed comedones affected nearly every single follicle, essentially involving the entire integument, even on sites not affected in common acne patients.

Due to the clinical picture and due to the fact that the working colleague of the patient had also started to develop multiple cysts in her face as well as gastrointestinal symptoms, the clinical diagnosis of chloracne was established. This diagnosis was confirmed by blood investigations which revealed highly elevated levels of 2,3,7,8-TCDD which were the highest levels ever been measured worldwide in adult patients (144.000 and 26.000 pg/blood lipids respectively). Histological investigation showed cystic dilation of the follicles filled with horny material and the absence of sebaceous glands, a condition which is reported to be a characteristic of chloracne (*6*).Laboratory findings in the more affected patient revealed leucocytosis and slightly elevated markers of inflammation. There was a transient elevation of serum lipids and liver enzymes, which partly could be attributed to the administration of isotretinoin, a vitamin A derivative, initiated in April 1998. Immunological (for details see report by K. Abraham in this issue), endocrine parameters and porphyrins in urine were normal as was neurological assessment and various apparative investigations. In the second patient routine laboratory parameters were within the normal range, as were immunological parameters (lymphocytes subpopulation of PBMCs) and porphyrine levels. However, during the observation period of 10 months an elevation of the thyroid stimulating hormone (TSH) could be documented, with normal peripheral thyroid hormone levels and thyroid gland morphology.In contrast to common acne, which has a different pathophysiology, there is currently no sufficient treatment available for patients with chloracne (*4*). Our first patient received isotretinoin systemically (in a dosage of 20mg bid) from April until December 1998, when a cumulative dosage of approximately 13g and the lack of clinical benefit made us to stop this medication. Apart from an optimistic report about the successful use of topical retinoid acid in mild cases (*7*), other authors reported oral isotretinoin to be ineffective in the treatment of chloracne (*4)*. Additionally, our patient intermittently was given systemic steroids to control her inflammatory skin condition. Recurrent deep inflammation of the cysts still requires surgical interventions. In the second patient the skin symptoms remained restricted to the malar crescent and the pre-and retroauricular area. Both patients received topical treatment in the face with retinoids and anti-inflammatory ointments applied by iontopheresis which had a satisfying effect.These two patients reported here are of particular medical interest in regard to long term effects of severe TCDD intoxication. Further follow-up will help to elucidate pathomechanisms of chloracne.

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