

Chloramphenicol Induced Vitiligo-like Depigmentation

Zekayi Kutlubay¹, MD, Burhan Engin¹, MD, Ronni Wolf², MD, Yalçın Tüzün¹, MD

Address: Departments of Dermatology, ¹Istanbul University Cerrahpaşa Medical Faculty, Turkey and

²Kaplan Medical Center, Rehovot, Israel

E-mail: zekayikutlubay@hotmail.com

* Corresponding Author: Dr. Zekayi Kutlubay. Department of Dermatology, İstanbul University Cerrahpaşa Medical Faculty, Fatih, İstanbul, 34098, Turkey

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Abstract

Observation: Vitiligo is a common acquired pigmentary skin disorder related to the selective loss of melanocytes. Aetiology of vitiligo is uncertain but seems to be dependent on the interaction of genetic, immunological and neurological factors. Its pathogenesis is still not understood. Chloramphenicol is one of the substances causing chemical leukoderma. We report an unusual case of topical chloramphenicol induced periocular vitiligo because of its rarity.

Introduction

Chemical leukoderma may appear identical to vitiligo and may have a similar anatomical distribution [1, 2, 3, 4]. The incubation period for exposure ranges from two weeks to approximately six months. Depigmentation is not always preceded by inflammation of the affected skin and the latter is certainly not a prerequisite. Most sources claim having difficulty in differentiating vitiligo from chemical leukoderma by light and electron microscopic examination [3, 4, 5, 6, 7, 8, 9, 10, 11].

Case Report

A fifteen-year old girl presented with periorbital depigmentation. She had applied chloramphenicol ointment daily for 1.5 years to the periorbital region, probably for treating chronic conjunctivitis and resultant dermatitis. Two months ago presenting vitiliginous macules appeared on the exact areas where she had applied the ointment. The patient was otherwise in good health, her past medical history was unremarkable except for recurrent

aphthous stomatitis and her familial history was noncontributory except for diabetes.

Investigation for atopy criteria revealed that cheilitis, and Dennie' s lines were present and cradle had been in the neonatal period. Thyroid function tests were within the normal range, hepatitis B antigen and antibody were negative, hematologic and biochemical examinations were normal, and no features of anemia and diabetes were detected. We



Figure 1. Periorbital depigmentation after prolonged use of chloramphenicol eye ointment

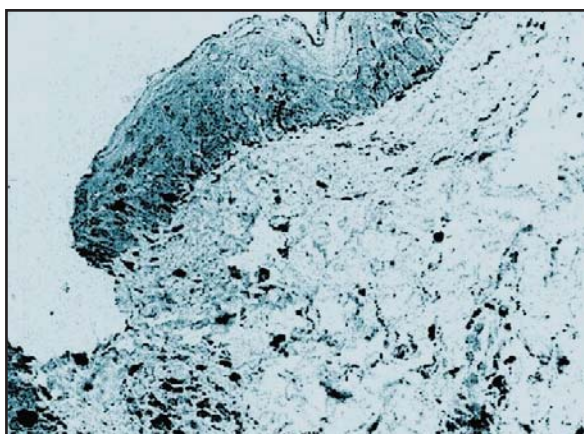


Figure 2. Absence of melanocytes at the basal layer (S-100 x 400)

could not find any sign of infection: the chest X-ray examination, erythrocyte sedimentation rate, blood smear and urinalysis were normal.

No pathology was noted on the ophthalmologic examination. The corneas and lenses were clear, anterior chambers were normal. There was no pathological evidence on fundus examination. Visual acuity was 10 (*Snellen* chart) with correction (myopia).

Dermatological examination: on the both periorbital regions there were depigmented macules within some normal colored skin areas. A few eyelashes showed poliosis (**Figure 1**).

Histopathological examination: Normal skin architecture was found on H+E staining. Fontana staining received diminished pigmentation in the basal layer. Absence of melanocytes was detected by the immunohistochemical stain S-100 (**Figure 2**).

Discussion

Evidence in favor of chemical leukoderma in our case includes onset of leukoderma associated with the application of chloramphenicol, halting the progression of leukoderma when the eye ointment was no longer used, no clinical evidence of vitiligo at other sites and no diseases known to be associated with vitiligo.

Korting, in his monography *The Skin and Eye* [12], and later *Cowan et al.* [13] and *Barnes* [14] pointed out that patients with idiopathic vitiligo often have ocular abnor-

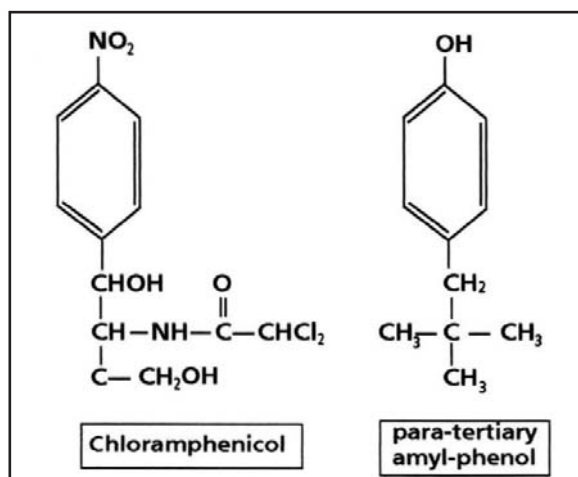


Figure 3. Similarity of chloramphenicol and para-tertiary amyl-phenol molecules

malities, such as hypopigmented spots of fundus, iris, eyebrows and lashes. Our patient did not show any eye abnormalities although some of her lashes were affected. Involvement of eyelashes, as in our patient, does not exclude chemically induced leukoderma, since this feature has been documented in several reports of chemically induced vitiligo, as well [2].

In conclusion, although we favor the diagnosis of chemical leukoderma, it is not possible to absolutely exclude vitiligo or Koebner-induced vitiligo. Other less likely diagnoses are postinflammatory hypopigmentation, scleroderma and lichen sclerosus et atrophicus, all of which we believe could be excluded on clinical and histopathological grounds.

Most known depigmenting agents are alkyl phenols with the alkyl, hydroxyl, or amino groups in the para position [15]. Chloramphenicol also possesses two groups (a nitro and a dichloro-N-hydroxy-hydroxyrnyethyl-ethyl group) in the para position of the benzene ring, and thus has structural similarities with other known depigmenting agents particularly, the para tertiary butyl phenol and para tertiary amyl phenol (**Figure 3**), two very potent depigmenting agents [15]. It is not surprising, therefore, that two additional cases of leukoderma induced by this agent have been reported by others [16, 17].

We believe that the number of such cases is higher than that reflected in the literature and that similar cases have been overlooked or misdiagnosed.

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