types of cabbage, 1/10 w/v in saline were coupled to CNBr-activated filter paper discs (5 µl per disc), which were used to determine IgE antibodies by RAST essentially as described earlier (3).

The patient's serum gave values of 7.6 and 6.1 PRU/ml (RAST class 3) (Phadebas RAST units, Pharmacia Diagnostics, Uppsala), respectively, to discs with *B. nigra* and *B. alba*. The results for normal sera and sera with high IgE levels (approximately 3000 kU/l) were less than 0.35 PRU/ml (negative RAST). RAST class 3 results were also obtained with rape-seeds while RAST was negative to cabbage. Thus, significant concentrations of IgE antibodies to mustard-related allergens were present in the patient's serum.

On the basis of the case history and the RAST results the patient was recommended to avoid all kinds of food that might contain mustard and she has had no further attack of urticaria.

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Red Scalp Hair Turning Dark-brown at 50 Years of Age

L. JUHLIN and J. P. ORTONNE

Department of Dermatology, Hôpital Pasteur, University of Nice, F-06031, Nice, France

Juhlin L, Ortonne JP. Red scalp hair turning dark-brown at 50 years of age. Acta Derm Venereol (Stockh) 1986; 66: 71–73.

We report on a man whose scalp hair was reddish since childhood but changed into dark-brown after the age of 50. His pubic hair and beard remained carrot coloured. A high level of arsenic in his scalp hair seemed to be a possible cause of the change from pheomelanogenesis to eumelanogenesis. *Key words: Hair colour; Arsenic; Pheomelanin.* (Received June 14, 1985.)

L. Juhlin, Department of Dermatology, University Hospital, S-751 85 Uppsala, Sweden.

Changes in hair colour in adults usually occur with age or may be induced by drugs such as antimalarials. In these cases there is a greying or lightening of the hair (1). It is known that red-haired children often become brown-haired, sandy-haired or auburn-haired as adults (2). Here we describe similar changes in an adult, a phenomenon that has not been reported in the literature before.

CASE REPORT

The patient is a 57-year-old man, who worked outdoors in vineyards, where he has been in contact with products which could contain arsenic. In the last 2 years squamous cell carcinomas of the face have been removed by surgical excision. Otherwise, he has been in good health.

Since childhood he was nicknamed 'the carrot' because of his carrot-coloured hair. His beard and pubic hair were of the same reddish colour. For the last 4 years he noticed that his scalp hair turned dark-brown, whereas genital and axillary hair remained red. The patient has not used any hair colour and only washes with ordinary soap.

Routine blood and urine tests were all normal. Arsenic levels in the urine were normal but increased in blood 0.39 μg/l (normal <0.06), nails 6.5 μg/g (normal <4.0), scalp hair 1.7 μg/g (normal <0.5) and pubic hair 0.12 μg/g (normal <0.05). Serum and hair levels of aluminium, lead and copper were within normal limits. For 4 weeks pubic and scalp hair were left on a microscope slide and exposed to sunlight. No colour changes were observed.

DISCUSSION

Visual differentiation of hair colour does not always reflect the melanogenesis type in human red hair. It is known that hair colour can be due to either pheomelanin or a mixture of phaeomelanin and eumelanin (3). The colour of brown or black hair results from a predominance of eumelanogenesis (4). The concentration of sulphated compounds, particularly the sulphydryl group (SH) within the pigment cell appears to be the prime element in deciding between eumelanogenesis (low level of SH) and phaeomelanogenesis (high level of SH) (5). Of the SH-compounds, reduced glutathione and cysteine are important, and the three enzymes glutamyltranspeptidase, glutathione reductase and glutathione peroxidase probably play key roles in regulating their levels (6).

The hair colour changes observed in our patient probably correspond to in vivo conversion of phaeomelanogenesis to eumelanogenesis. Two environmental factors may be implicated in this phenomenon: overexposure to sun light and chronic arsenic intoxication or both. A change of hair colour from red to black-brown in 'phaeomelanic' individuals after chronic heavy sun exposure has not been observed before although UV-radiation has been shown to decrease the level of both glutathione reductase and reduced glutathione (7). It would appear that arsenic is the most probable cause of the change of hair colour in our patient. Arsenic binds easily to thiol groups including those of glutathione and the decrease in the level of reduced glutathione by As^{3+} derivatives is proportional to the logarithm of the arsenic dose (8).

One may suggest that the high level of arsenic in the scalp hair of our patient induced a strong decrease in the level of reduced glutathione, resulting in a switch from phaeomelanogenesis to eumelanogenesis. In the axillary and pubic hair the levels of arsenic were found to be 10 times lower and were not sufficient to modify phaeomelanogenesis. We have no explanation for the differences in the arsenic content between scalp and pubic hair but it is well established that a similar difference exists in normal subjects without arsenic intoxication (9). The influence of arsenic on melanogenesis therefore seems to be worth further study.

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Long Term Plasma Exchange Therapy in Bullous Pemphigoid

B. GUILLOT,[1] D. DONADIO,[2] J. J. GUILHOU[1] and J. MEYNADIER[1]

[1]*Service de Dermatologie, Hôpital Saint-Charles and* [2]*Service des Maladies du Sang, Hôpital Saint-Eloi, Montpellier, France*

Guillot B, Donadio D, Guilhou JJ, Meynadier J. Long term plasma exchange therapy in bullous pemphigoid. Acta Derm Venereol (Stockh) 1986; 66: 73–75.

Long term plasma exchanges associated with corticosteroids (11 patients) were compared with corticotherapy alone (10 patients) for the treatment of bullous pemphigoid. The patients having long term plasma exchanges showed a lower rate of relapses at six months and needed less corticosteroids. *Key words: Maintenance therapy; Corticosteroid therapy; Early relapse.* (Received April 30, 1985.)

B. Guillot, Service de Dermatologie, Hôpital Saint-Charles, F-34059 Montpellier Cedex, France.

Bullous pemphigoid is a blistering autoimmune skin disease characterized by linear deposits of immunoglobulins and complement components along the basement membrane zone (BMZ). Circulating antibodies against BMZ are present in 2 out of 3 patients and circulating immune complexes are often detected. The usual therapy for bullous pemphigoid is high dosage of corticosteroids associated with immunosuppressive drugs in the more severe forms of the disease. Adverse reactions to these treatments are serious. Recently in a few open studies (1, 2, 3, 4, 5), plasma exchanges (P.E.) appeared to be effective in the treatment of bullous pemphigoid. A multicentric randomized trial suggest that plasma exchanges allows a substantial saving of corticosteroids in the management of the disease (6). However the rôle of long term plasma exchanges in bullous pemphigoid is unknown. We report an open study of 18 patients treated with or without periodical P.E. given after a remission (defined as more than 6 weeks) was obtained.

PATIENTS AND METHODS

Eighteen patients were included in this study, 5 women and 13 men, 46 to 88 years old. The diagnosis of bullous pemphigoid was confirmed by histopathology and immunological studies. Circulating antibody against BMZ was present in 11 patients.

All patients in this study were in remission after the first treatment (corticosteroids alone, corticosteroids and plasma exchanges, or plasma exchanges alone). The corticosteroids dose (prednisolone) was reduced every 15th day by 15% approximately of the previous dosage.