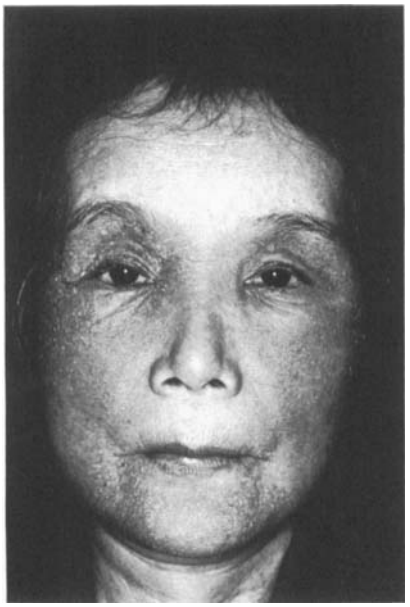

Fig. 1. Erythematous rash on the face and neck.

tosensitivity with thrombocytopenia has not been reported. The mechanism by which photosensitivity and thrombocytopenia are produced is unknown. The possibility that the fall in platelet count represented an idiopathic thrombocytopenia cannot be dismissed [5], however, the temporal relationship between the initiation of the drug and the onset of a skin rash and thrombocytopenia suggests more than a chance association.

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Growth of Eyebrow After Alpha Interferon Administration

To the Editor: Administration of alpha-interferon (α-IFN) is increasingly used in treatment of viral hepatitis and hematological malignancies including chronic myelogeneous leukemia, multiple myeloma, and hairy cell leukemia. Adverse effects of α-IFN include flu-like symptoms and gastrointestinal symptoms. We encountered the rare side effect of α-IFN in a patient with growth of the eyebrows.

A 57-year-old male was diagnosed with multiple myeloma in March 1933. Radiographic examination revealed punched-out lesions of the skull, tumor of the sternum, fracture of the ribs and throacic veretebrae. Hematological examination revealed: Hb 6.3 g/dl, platelet $8.3 \times 10^4/\mu l$, WBC 4,300/μl. Biochemistry revealed: total protein 14.7 g/dl, albumin 2.8 g/dl, IgA 10,980 mg/dl, κ. Bone marrow aspiration revealed 100% of myeloma cells. The patient was in stage III. He was treated with three courses of VAD (vincristine, adriamycin, and dexamethasone) chemotherapy with simultaneous administration of interferon alpha-2b, 300 MU/day. Radiation therapy was performed for the sternum tumor. Bone pain decreased, IgA level decreased to 1,360 mg/dl, and bone marrow plasma cells decreased to 7.6%. The patient was discharged and daily administration of α-IFN was continued. The patient was hospitalized again in September 1994 since IgA level increased to 5,000 mg/dl and bone marrow aspiration revealed 27.6% myeloma cells. Three more courses of VAD chemotherapy were administered and α-IFN was continued thereafter.

Growth of the eyebrows was noted approximately 3 months after administation of α-IFN was started. The eyebrow hair fell out soon after each course of chemotherapy, but grew again soon after administration of α-IFN, and maximum length was approximately 10 cm (Fig. 1).

This is the first reported case of eyebrow growth due to α-IFN. Pathogenesis of eyebrow growth due to α-IFN is unknown. There have been a few reports of the eye lash growth [1,2]. In a case, cimetidine, administered with α-IFN, was considered the causative agent [1]. However, in our case, no other drugs were co-administered with α-IFN. There is also a report of the growth of scalp hair [2]. In our case, only eyebrow growth was observed.

In a case of Crow-Fukas syndrome due to plasma cell dysclasia, manifestation of hirsutism was observed [3]. However, in our case, multiple myeloma was well controlled.

Androgens had been reported to have paradoxically different effects on human hair follicles depending on their body sites [4]. Androgens stimulate hair growth such as beard and pubis, have little effects on protective hair such as those of the eye lash, but can cause regression or balding of the scalp hair. Our case might not be related to androgen metabolism since the eyebrows grew.

The immune system of the hair follicle may play a role in hair growth. Alpha-IFN not only acts on T-cells, but also on antigen presenting cells such as macrophages, and these cells may regulate hair growth via the release of cytokines such as γ-IFN and tumor necrosis factor [5]. There is additional evidence that the alteration of the immune system of the hair follicle affects hair growth. Defective monocyte/macrophage function, which significantly diminishes production of tumor necrosis factor α by these cells, has been demonstrated in patients with alopecia universalis [6]. One of the well-established side effects of the immunosuppressive agent cyclosporin A is hirsutism [7], and the primary action of this drug is interference with the production of cytokines such as interleukin-2 and

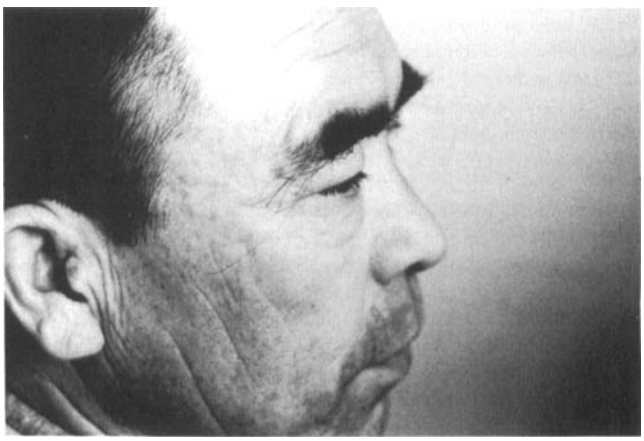

Fig. 1. Growth of eyebrow in a patient receiving alpha interferon.

γ-IFN. The rare effect of α-IFN on the growth of the eyebrow in the present patient might be due to the altered immune function of the follicle.

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Adult T-Cell Leukemia Diagnosed After 22 Years

To the Editor: Adult T-cell leukemia (ATL), first proposed as a new disease entity in 1977 [1], is now known to be caused by human T-lymphotropic virus type I (HTLV-I) [2,3]. Presumably, we failed to recognize ATL for many years before 1977 in Japan where HTLV-I is endemic. How many years back we can trace ATL cases is an interesting subject. In 1973, we treated a 38-year-old Japanese woman who presented with characteristic clinical features of ATL such as abnormal T-lymphocytosis with indented or lobulated nuclei, lymphadenopathy, hepato-splenomegaly, and rapidly fetal outcome with histologically proven cytomegalovirus pneumonia. At that time, we were puzzled by the disease. After 22 years, we were able to detect HTLV-I proviral sequences in DNA extracted from her unstained blood smears by polymerase chain reaction using primers specific for the pX region (Fig. 1) [4]. Although her serum was not available for antibody testing, the result strongly suggests that our patient had ATL and that this technique is worth trying in archival tissue samples from patients suspected to have had ATL.

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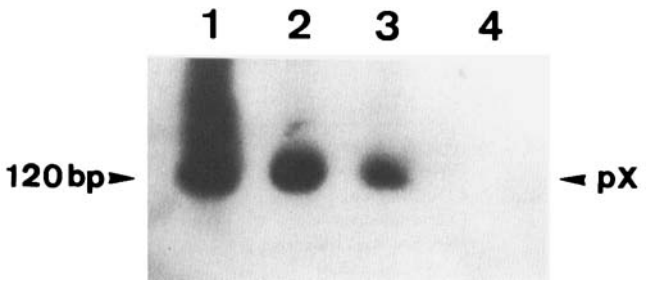

Fig. 1. Southern blot analysis of HTLV-I pX sequences amplified from peripheral blood smears by polymerase chain reaction. Lane 1, ATL patient; lane 2, present patient; lane 3, HTLV-I carrier; lane 4, H_2O.

Unscreened Transfusion Related Human Immunodeficiency Virus Type-I Infection Amongst Indian Thalassemic Children

To the Editor: Studies in India have shown that the two important population groups at risk for human immunodeficiency virus type I (HIV-I) infection are heterosexually promiscuous persons and paid blood donors [1]. The latter group give blood on payment and constitute the majority of donors in most blood banks. In addition, paid donors are from the poor socio-economic strata who usually indulge in heterosexual promiscuity. Rates of seropositivity are therefore alarmingly high in paid donors, up to 75% compared to a rate of 0.34% in other donors [1]. Transfusion of infected blood has up to 90% risk of transmission of the HIV-I infection [2]. In India, mandatory screening of all donated blood came into practice in March 1989. However, because stringent control is not always implemented, infected blood transfusion practices appear to continue with consequent fatalities.

The thalassemic clinic at Sanjay Ghandi Hospital, Manipur is a major transfusion centre in India. Here, many thalassemic children continue to receive unscreened blood from paid donors. Systematic screening for HIV-I infection of all multitransfused thalassemic children was therefore undertaken from August 1992 til November 1994 while investigating the obstetric outcome of their mothers. Preliminary results of the incidence and clinical manifestation of HIV-I infection in this high risk pediatric population are presented.

The sera of 406 multitransfused children with various types of thalssemia were screened for HIV-I antibodies by competetive ELISA (Wellcozyme Recombinant, Wellcome Diagnostic, UK). Confirmatory Western blot was performed on all ELISA positive sera.

Immunologic status of the seropositive children was evaluated by

1. Absolute lymphocyte count