INTRODUCTION

Hypertrichosis of eyebrows and eyelashes is often considered synonymously due to their anatomic, physiologic, and embryologic similarities. An objective diagnosis of eyelash trichomegaly may be made if eyelashes measure more than 12 mm in length. Recent reports have also considered features such as curling, thickening, and increased pigmentation of eyelashes to denote trichomegaly.

The term “trichomegaly” was coined by Gray in 1944 to describe increased length of eyelashes while reporting a case of lymphoma. Eyelash trichomegaly may be presenting the feature of various congenital and acquired conditions. It may also be induced by topical as well as systemic drugs. The pathogenesis of trichomegaly has not been elucidated.

We hereby report a rare case of systemic lupus erythematosus (SLE) in association with eyelash trichomegaly, “lupus hair,” and diffuse hair loss. Written informed consent of the parents was taken.

CASE REPORT

An Indian girl aged 16 years presented with complaints of fatigue, malaise, joint pains, reduced appetite, sunlight sensitivity, and intermittent low-grade fever of 1½ years duration. She also complained of gradual, diffuse hair loss over the scalp and an increase in length with curling of eyelashes of 1 year duration. She had to trim her eyelashes twice a month. She was not taking any oral or topical medication other than oral prednisolone in a dose of 20 mg/day intermittently for the past 4 months. None of her family members had hypertrichosis of the eyebrows or eyelashes.

General physical and systemic examination did not reveal any abnormalities. Mild erythema was observed in the paranasal area. Mild diffuse alopecia scalp, coarse dry, unruly, broken “lupus hair” along the hairline of the scalp, and trichomegaly of eyelashes which were nearly 15 mm in length were also observed [Figure 1]. Routine hematological examination revealed Hb 10.8 g/dL, leukopenia with total leukocyte count 3200/mm³, differential leukocyte count (P, L, M, E), erythrocyte sedimentation rate

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How to cite this article: Dalal A, Sharma S, Kumar A, Sharma N. Eyelash trichomegaly: A rare presenting feature of systemic lupus erythematosus. Int J Trichol 2017;9:79-81.
46 mm/h, and thrombocytopenia with platelet count 90,000/mm³ while routine urine test did not reveal any proteins or casts. Antinuclear antibodies and anti-dsDNA antibodies were positive. A diagnosis of SLE was made and the patient referred to the rheumatologist for further evaluation and management.

**DISCUSSION**

Eyelashes are the first terminal hair to appear during embryological development at 12 weeks of gestation. With a short anagen phase of nearly 30 days and a relatively long telogen phase of 4–5 months, their growth cycle is completed in 5–6 months. The length of eyelashes of upper eyelids ranges from 8 to 12 mm. Growth of eyelashes to a length >12 mm or thickening, increased pigmentation, or curling of hair is termed trichomegaly. Eyelash trichomegaly may be a benign, solitary finding in an otherwise healthy individual, a part of a congenital syndrome such as the Cornelia de Lange syndrome, a sign of an acquired condition such as HIV infection or induced by topical, or systemic drugs [Table 1].  Although systemic drugs such as cyclosporine, epidermal growth factor receptor (EGFR) inhibitors, and interferon alpha may induce eyelash trichomegaly, systemic corticosteroids have not been associated with trichomegaly. On the contrary, systemic steroid administration has been associated with iatrogenic hirsutism with the appearance of terminal hair in the beard area of the face.

SLE is a multisystem autoimmune inflammatory disease with fibrinoid changes and vascular abnormalities. Hair changes are frequently observed in SLE. Recently, nonscarring alopecia has been included in the classification criteria for SLE. Hair changes include diffuse alopecia occurring in up to 50% and unruly, short, broken-off “lupus hair” in 30% of patients. Eyelash trichomegaly associated with SLE is a rare finding. To the best of our knowledge, only two cases

| Conditions | Common | Uncommon | Rare |
|------------|--------|----------|------|
| Congenital | Cornelia de Lange syndrome (Synophrys, facial and musculoskeletal anomalies, small stature, developmental delay) Oliver–McFarlane syndrome (Pigmentary retinal degeneration, dwarfism, mental retardation) | Congenital heart disease (Various congenital heart defects, including tetralogy of Fallot) Familial trichomegaly (Eyelash trichomegaly in absence of other abnormalities) Hermansky–Pudlak syndrome (Oculocutaneous albinism, platelet storage pool deficiency, lysosomal accumulation of ceroid lipofuscin) | Aghaei–Dastgheib syndrome (Generalized hypertrichosis, bilateral nipple retraction, accessory nipple) Cone–rod dystrophy (Cone rod amaurosis, hypertrichosis) Goldstein–Hutt syndrome (Cataracts, hereditary spherocytosis) Phylloid hypomelanosis (Hypopigmented leaf-like macules, mosaic trisomy 13, cerebral, ocular and skeletal defects) |
| Acquired | Atopic dermatitis/allergic disease HIV Uveitis Vernal keratoconjunctivitis | | Alopecia areata Cancer (metastatic renal adenocarcinoma) Dermatomyositis Systemic lupus erythematosus |
| Drugs | Epidermal growth factor receptor inhibitors Monoclonal antibodies: Cetuximab Tyrosine kinase inhibitors: Erlotinib Prostaglandin analogs Bimatoprost (frequent) Latanoprost (frequent) Travoprost (frequent) | Panitumumab Gefitinib Interferon alpha | Cyclosporine Tacrolimus Topiramate Zidovudine |

*The following terms are used to define the occurrence of eyelash trichomegaly: rare (fewer than five cases reported); uncommon (multiple cases reported); and common (well-known association proven in clinical studies).*
of eyelash trichomegaly in association with SLE have been reported till date.[7] In our patient, diffuse alopecia and “lupus hair” were observed in addition to eyelash trichomegaly.

The pathogenesis of trichomegaly has not been elucidated because human eyelash hair follicles in contrast to scalp hair follicles are not available for histology or organ culture. At the molecular level, it has been postulated that the nuclear factor of activated T-cells (NFAT) blocks stem cell activation in the bulge and suprabulbar region of the eyelash hair follicles. Inactivation of NFAT may cause trichomegaly. It is also postulated that EGFR signaling acts as a “brake” on the growth cycling of the eyelash hair follicles. Inhibition of EGFR may result in trichomegaly.[8] We are reporting a rare case of SLE who presented to us with trichomegaly of the eyelashes along with “lupus hair” and diffuse alopecia scalp. Inhibition of EGFR signaling in the eyelash hair follicles due to the disease process may be responsible for the increase in length of the eyelashes in our patient.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal.

The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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