The trichoscopic features of hair shaft anomalies induced by epidermal growth factor receptor inhibitors: A case series

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Background: Although the clinical hair changes that occur under treatment with epidermal growth factor receptor inhibitors (EGFRIs) are documented, their trichoscopic features have not been reported.

Objective: To evaluate the trichoscopic findings in scalp and facial hair, induced by EGFRI treatment.

Methods: Patients treated with EGFRIs at a tertiary oncodermatology clinic in 2015 through 2017 were evaluated for macroscopic and trichoscopic changes.

Results: The cohort included 23 patients (13 women; median age, 68 years) treated with EGFRIs for an average of 13 months (range, 2-40 months). Macroscopically, 18 patients (78%) had dry, lusterless, coarse, kinky, brittle scalp hair, and 17 (74%) had trichomegaly of the eyebrows/eyelashes. Trichoscopic findings were of hair shaft anomalies including pili torti, affecting scalp hair in 20 patients (87%), eyebrows in 6 (26%), and eyelashes in 8 (50%), and asymmetric hyperpigmented fusiform widening of hair scalp in 3 (13%), eyebrows in 10 (43%), and eyelashes in 4 (25%). Dermoscopic findings of the peri- and interfollicular skin were scale, whitish erythematous structureless areas, and branching vessels.

Limitations: Lack of trichoscopic-histologic correlation, lack of baseline examination.

Conclusion: The trichoscopic correlates of the macroscopic hair changes under EFGRI treatment include pili torti, and asymmetric hyperpigmented fusiform widening, with dermoscopic cutaneous manifestations of scale, whitish erythematous structureless areas, and branching vessels. (J Am Acad Dermatol 2021;85:1178-84.)

Key words: asymmetric hyperpigmented fusiform widening; dermoscopy; epidermal growth factor receptor inhibitor; hair; pili torti; trichoscopy.

Epidermal growth factor (EGF) receptor (EGFR) is a member of the subfamily of receptor tyrosine kinases that plays important roles in cell regulation, migration, and differentiation. Overexpression of EGFR or its ligands or activating mutations in the EGFR signaling pathway may lead to the formation of epithelial neoplasms. This process is characteristic of a large number of cancers in various tissues and has prompted the development and steadily growing therapeutic use of EGFR inhibitors (EGFRIs). There are 2 main types of EGFRIs: monoclonal antibodies (mAbs) that block the extracellular domain of the receptor and small-molecule tyrosine kinase inhibitors (TKIs) that block the intracytoplasmic adenosine triphosphate–binding site of the receptor.[1,2]

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EGFR expression is normally high in proliferating basal keratinocytes in the epidermis and decreases as the keratinocytes migrate to the suprabasal layers.[3] In addition, they are expressed on the outer layer of the hair follicle and on sebaceous and eccrine sweat glands.

In patients with cancer, treatment with EGFRIs, either mAbs or TKIs, is commonly associated with adverse effects in skin and hair, namely, papulopustular rash, mainly of the upper trunk and face (60%-90%); dry and itchy skin (at least in 12%-16%); microbial infections (38%-70%), pruritus; and paronychial inflammation.[1,4]

The reported rates of macroscopic changes in hair in patients treated with EGFRIs range from 6% to 21%. Onset of these hair changes is variable, from as early as the second month after starting treatment to months later.[1-5] Manifestations include kinky, coarse, lusterless, and more brittle hair on the scalp and extremities; trichomegaly with rigid, thickened, and curling eyelashes and eyebrows; and hypertrichosis of the face.[1-5] Alopecia may also develop, usually manifesting as an androgenetic hair loss pattern with the potential to progress to scarring alopecia.[6] The pathogenesis of these hair clinical phenotypes has been investigated, indicating in EGFR-deficient skin models that, among other findings,[2-4,7] EGFR controls transcription factor expression in the innermost epithelial hair lineages, which is required for hair shaft differentiation.[7]

Although the clinical effects of treatment with EGFRIs on hair have been well documented, there are only a few reports in the literature of their corresponding trichoscopic (hair and scalp dermoscopy) features.[6,8-10]

The aim of the present study was to describe the trichoscopic hair findings associated with the known macroscopic changes induced by EGFRIs.

CAPSULE SUMMARY

- Macroscopic hair changes under epidermal growth factor receptor inhibitors include coarse, lusterless hair and trichomegaly. We found correlating trichoscopic hair shaft anomalies, which included pili torti and asymmetric hyperpigmented fusiform widening, as well as dermoscopic cutaneous scale, whitish erythematous structureless areas, and branching vessels.
- These observations may improve our understanding of the role of epidermal growth factor receptor in hair physiology.

METHODS

Setting and patients

Inclusion criteria for the study were patients treated with EGFRIs (mAbs or TKIs) for at least 2 months, for any type of cancer, with macroscopic changes of scalp and/or eyebrow and/or eyelash hair, attributed and typical to treatment with EGFRIs, including kinky, coarse, lusterless, and more brittle hair of the scalp and/or trichomegaly with rigid, thickened, and curling eyelashes and/or eyebrows. All patients were examined at the Rabin Medical Center tertiary oncodermatology clinic between May 2015 and July 2017.

Exclusion criteria were patients treated with EGFRIs for less than 2 months and patients not treated with EGFRI at the time of evaluation.

All patients were referred from the Davidoff Cancer Center for treatment of the cutaneous adverse effects of EGFRIs.

The clinical and trichoscopic features of the scalp hair, eyebrows, and eyelashes were documented, as were the clinical and dermoscopic manifestations of the perifollicular and interfollicular skin in these areas. Pictures of the macroscopic and dermoscopic findings in the areas most prominently affected were obtained for each patient.

The medical records of each patient were reviewed for demographic data (age, sex), type of malignancy being treated with EGFRI, type of EGFRI administered, and duration of EGFRI use.

The study was approved by the local institutional review committee of Rabin Medical Center.

Statistical analysis

Categorical variables were compared between groups by Fisher exact and chi-square tests. For continuous variables, statistical significance was calculated by the nonparametric Wilcoxon measure.

A 2-tailed *P* value of less than .05 was considered statistically significant. The statistical analysis was conducted with SAS, version 9.4.

RESULTS

Twenty-three patients met the study criteria. Table I summarizes their clinical and trichoscopic characteristics. There were 13 women and 10 men with a median age of 68 years (range, 38-87). All were white. Eleven patients were treated with an mAb (cetuximab, 7; panitumumab, 4) and 12 with a TKI (afatinib, 5; erlotinib, 5; gefitinib, 2). Tumors included lung adenocarcinoma in 9 patients, colon/rectal carcinoma in 9, squamous cell carcinoma in 4 (1 each in the lung, vocal cord and

Abbreviations used:

EGF: epidermal growth factor
EGFR: epidermal growth factor receptor
EGFRI: EGFR inhibitor
mAb: monoclonal antibody
TKI: tyrosine kinase inhibitors

lung, esophagus, and nasal cavity), and polycythemia vera in 1. The average time from starting EGFRI treatment was 13 months (range, 2-40 months).

All patients had macroscopic features of scalp hair and/or eyebrows and/or eyelashes typically observed under treatment with EGFRIs.[1-5] The scalp hair was dry, lusterless, coarse, kinky, fragile, and brittle in 18 patients (78%) and normal looking in 5. Seventeen patients (74%), including 4 of 5 with normal scalp hair, had trichomegaly of the eyebrows and eyelashes with long, curly, thick, and rigid hairs. The remaining patient with normal-looking scalp hair had trichomegaly only of the eyebrows. Ten patients (43%) had hypertrichosis of the face, of whom 9 were women (69% of all women patients).

On trichoscopic evaluation of the scalp hair, pili torti was found in 20 patients (87%), with hair shafts with sharp bending at irregular intervals and flattened hair shaft with regular twists at irregular intervals (Fig 1, *A* and *B, arrows*); 3 (13%) of them also had a different type of hair shaft anomaly, displaying asymmetric hyperpigmented fusiform widening of the hair shaft (Fig 1, *B, circles*). Clinically, 18 had coarse scalp hair (Fig 1, *A*), and 2 had normal hair. The 3 patients without pili torti on the scalp had clinically normal-appearing hair.

Evaluation of the eyebrow hairs yielded the same asymmetric hyperpigmented fusiform widening of hair shaft in 10 patients (43%) (Fig 2) and pili torti in 6 (26%), with 3 patients displaying both anomalies simultaneously. Of the patients with trichoscopic changes in eyebrow hair, 10 had eyebrow trichomegaly, and 3 had clinically normal-looking eyebrows.

Trichoscopic findings in eyelashes included pili torti in 8 patients (50%; information was available for 16 patients). Four patients had intermittently hyperpigmented fusiform widening of hair shaft; 2 also had pili torti. Seven patients with trichoscopic changes in eyelash hair had eyelash trichomegaly, and 1 had clinically normal-looking eyelashes.

Overall, no significant differences were found regarding the trichoscopic findings and their prevalence in the hair of the scalp, eyebrows, or eyelashes, or in all 3 compartments together, between patients treated with mAbs or TKIs.

No significant correlation was found between the trichoscopic findings in the scalp, eyebrows, or eyelashes and the duration of EGFRI treatment.

All 23 patients with EGFRI-induced hair changes had clinical macroscopic features resembling seborrheic dermatitis in the skin of the scalp, eyebrows, and eyelashes in the interfollicular and perifollicular areas, including mild erythema with white and yellow scale. Dermoscopic features of these changes appeared as adherent whitish and yellowish scale of the skin and base of the hairs as well as perifollicular and interfollicular whitish-erythematous structureless areas and branching vessels (Fig 3).

DISCUSSION

Patients treated with EGFRIs who develop clinical hair changes manifest specific trichoscopic hair shaft anomalies, mainly pili torti.

Most of the patients in our study had dry, lusterless, coarse, kinky, fragile, and brittle scalp hair; trichomegaly (with long, curly, thick, and rigid hairs) of the eyebrows and/or eyelashes; and, less commonly, hypertrichosis, all of which are typical macroscopic findings reported in the literature.[1-5]

The main trichoscopic findings in the study group were hair shaft anomalies including pili torti of the scalp hair (87% of patients), eyebrows (26%), and/or eyelashes (50%) and asymmetric hyperpigmented fusiform widening of the hair shaft in scalp hair (13%), eyebrows (43%), and eyelashes (25%). In partial accordance with our findings, Pirmez et al[8] described 2 patients treated with erlotinib for 1 and 4 years who had trichoscopic scalp hair features of black dots, pili torti, and broken hairs. Fukui et al[6] described a patient with trichoscopic features of follicular keratotic plugging, milky red areas, white patches, disordered hair shaft, and tapering hair at 11 months after starting treatment with gefitinib. Ena et al[9] reported trichoscopic findings of tufted hairs after 4 months of lapatinib therapy.

A classification of hair shaft abnormalities in trichoscopy was proposed by Rudnicka et al.[11,12] It distinguishes the following groups of hair shaft features observed by trichoscopy: (1) hair shafts with fractures, (2) hair narrowings, (3) hairs with node-like structures, (4) curls and twists, (5) bands, and (6) short hairs.

Pili torti (Latin: *pili*, hair and *torti*, twisted) is characterized by hair that microscopically appears flattened at irregular intervals and is twisted 180° along its axis. Clinically, it presents as dry, lusterless,

Table I. Characteristics of patients treated with epidermal growth factor receptor inhibitors

Patient no.	Age, y/sex	Type of malignancy	Drug used	Duration of treatment, mo	Macroscopic hair changes			Trichoscopic changes			Facial hypertrichosis (yes/no)
					Scalp	Eyebrows	Eyelashes	Scalp	Eyebrows	Eyelashes	
Patients treated with monoclonal antibodies											
1	45/M	Colon carcinoma	Cetuximab	10	Normal	Trichomegaly	Trichomegaly	Normal	AHFW	Pili torti	Yes
2	62/F	Esophagus SCC		2	Normal	Trichomegaly	Trichomegaly	Normal	Normal	Normal	No
3	68/M	Rectal carcinoma		5.5	Typical*	Trichomegaly	Normal	Pili torti	Normal	AHFW	No
4	69/M	Vocal cord and lung SCC		6	Typical*	Trichomegaly	Trichomegaly	Pili torti, AHFW	Pili torti, AHFW	Pili torti	No
5	73/M	Colon carcinoma		20	Typical*	Trichomegaly	Trichomegaly	Pili torti	AHFW	Pili torti	No
6	75/M	Colon carcinoma		4	Typical*	Trichomegaly	Trichomegaly	Pili torti	AHFW	NA	No
7	84/M	Nasal cavity invasive SCC		40	Typical*	Trichomegaly	Trichomegaly	Pili torti	Normal	NA	No
8	52/F	Rectal carcinoma	Panitumumab	7	Typical*	Trichomegaly	Trichomegaly	Pili torti	Normal	Pili torti AHFW	Yes
9	52/F	Colon carcinoma		9.5	Typical*	Trichomegaly	Trichomegaly	Pili torti	AHFW	Pili torti	Yes
10	66/F	Colon carcinoma		6	Typical*	Trichomegaly	Trichomegaly	Pili torti	Pili torti	Pili torti	Yes
11	73/F	Rectal carcinoma		5	Typical*	Normal	Trichomegaly	Pili torti, AHFW	Pili torti	Normal	Yes
Patients treated with small molecule tyrosine kinase inhibitors											
12	38/F	Lung adenocarcinoma	Afatinib	4	Typical*	Trichomegaly	Trichomegaly	Pili torti, AHFW	AHFW	AHFW	Yes
13	63/F	Lung adenocarcinoma		5	Typical*	Trichomegaly	Trichomegaly	Pili torti	Pili torti, AHFW	Pili torti	Yes
14	63/M	Lung adenocarcinoma		6	Typical*	Trichomegaly	Trichomegaly	Pili torti	Normal	NA	No
15	74/M	Lung SCC		5.5	Normal	Trichomegaly	Trichomegaly	Pili torti	AHFW	NA	No
16	87/M	Lung adenocarcinoma		10	Typical*	Trichomegaly	Trichomegaly	Pili torti	Pili torti	NA	No
17	58/F	Lung adenocarcinoma	Erlotinib	40	Typical*	Trichomegaly	Trichomegaly	Pili torti	AHFW	NA	No
18	68/M	Polycythemia vera		25	Typical*	Normal	Normal	Pili torti	Normal	Normal	No
19	72/F	Lung adenocarcinoma		16	Normal	Trichomegaly	Normal	Normal	Normal	Normal	Yes
20	79/F	Lung adenocarcinoma		18.5	Typical*	Trichomegaly	Trichomegaly	Pili torti	Normal	Pili torti	Yes
21	86/F	Lung adenocarcinoma		24.5	Typical*	Normal	Trichomegaly	Pili torti	Pili torti, AHFW	Pili torti, AHFW	Yes
22	79/F	Colon carcinoma	Gefitinib	7	Normal	Trichomegaly	Trichomegaly	Pili torti	Normal	Normal	No
23	87/F	Lung adenocarcinoma		9.5	Typical*	Normal	NA	Pili torti	Normal	NA	No

AHFW, Asymmetric hyperpigmented fusiform widening of the hair shaft; *F*, female; *M*, male; *NA*, not available, *SCC*, squamous cell carcinoma.
*Typical macroscopic manifestations include kinky, lusterless, and brittle hair.

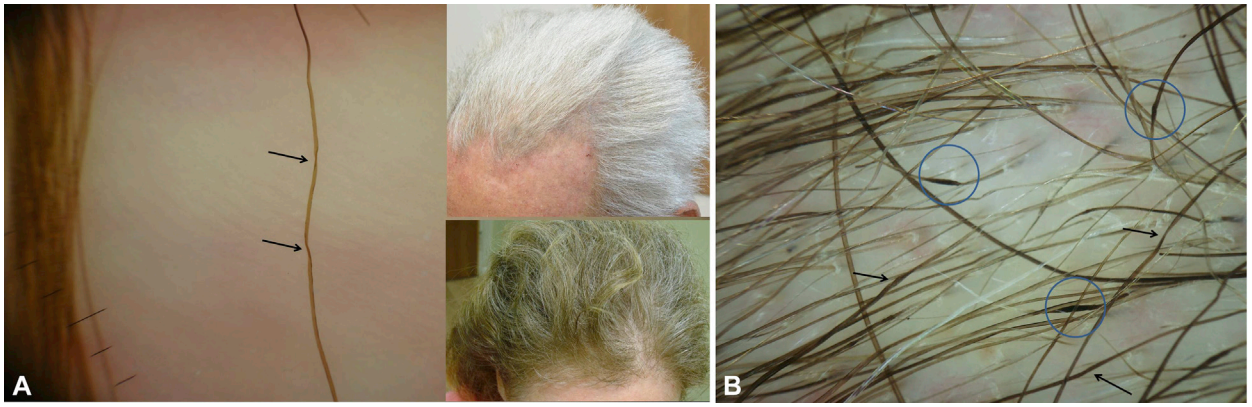

Fig 1. The clinical and trichoscopic changes of the scalp hair of patients treated with EGFRIs. **A**, Trichoscopic changes of the hair shaft with bending at irregular intervals characteristic to pili torti (black arrows). Macroscopic changes of dry, lusterless, coarse hair. **B**, Trichoscopic changes of asymmetric hyperpigmented fusiform widening of hair shaft (blue circles) and pili torti (black arrows).

Fig 2. The clinical and trichoscopic changes of the eyebrows, including trichoscopic changes of asymmetric hyperpigmented fusiform widening of hair shaft (blue circles), some with bending at irregular intervals characteristic of pili torti. Macroscopic changes of trichomegaly of the eyebrows and eyelashes with thick and rigid hairs.

coarse, fragile, and brittle hair, as in our patients. It is usually found as part of a number of well-known congenital syndromes. Acquired pili torti is less common and has been linked to anorexia nervosa, biotin deficiency, oral retinoid therapy, graft-versus-host disease, and residual hairs in cicatricial alopecia.[13,14] The wide range of disorders associated with pili torti suggests that different pathophysiologic mechanisms are responsible.[13] In congenital pili torti, alterations of the inner root sheath may lead to the abnormal molding and twisting of the hair shaft. In acquired pili torti, perifollicular fibrosis was assumed to cause rotational forces that distort the hair shaft follicle.[14]

It is generally recommended that people with pili torti try to avoid trauma to the hair. General suggestions include sleeping on a satin pillowcase, avoiding excessive grooming, braiding, heat treatments, dying, and coloring; reducing exposure to sunlight; using gentle shampoos; avoiding use of a hair dryer (or using it on the cool setting); and avoiding oral retinoids.[15,16]

We reported on a novel trichoscopic hair shaft finding, characterized by asymmetric hyperpigmented fusiform widening, which to our knowledge has not been reported in other acquired or congenital skin disorders. This hair shaft anomaly is a specific iatrogenic pathognomonic finding attributed to treatment with EGFRI. We did not observe the 180° twist along the shaft axis at the areas of this fusiform pigmentation, and therefore we assume that this phenomenon is not a form of exaggerated pili torti.

Although there were a few points with atypical brush-like ends of hair (fractured and frayed ends), we did not observe clear full findings indicating that this phenomenon is in the spectrum of trichorrhexis nodosa (no white nodes along the hair shaft with breaking at these points were observed).[11]

EGFR ligands, including EGF and transforming growth factor alpha, play a critical role during the hair cycle, triggering the anagen (growth) and catagen (transition) phases (EGFR mediates the termination of the anagen phase).[2,17] Studies have shown that in mice harboring a disruption of the EGFR allele, hair follicles failed to enter the catagen phase and remained in an aberrant anagen state, leading to thinning or loss of the outer and inner root sheaths, with fibrosis and an inflammatory perifollicular infiltrate.[2,3] Macroscopically, these changes manifested as short wavy hair and curling

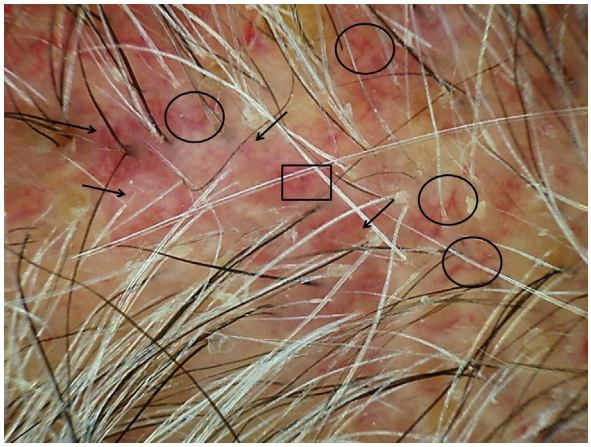

Fig 3. Dermoscopic changes of the skin of the scalp, consisting of adherent whitish and yellowish scale, whitish erythematous structureless areas (black arrows), branching vessels (black circles), and a few dotted vessels (black rectangle).

of whiskers, eventually followed by hair follicle degeneration and alopecia.[3,4]

Recently, Amberg et al[7] reported that EGFR signaling affects spatial layering during hair follicle growth and differentiation. They suggest that first, EGFR promotes cell proliferation and ensures DNA integrity in highly proliferative epidermal compartments, such as interfollicular epidermis, outer root sheath, and matrix. Upon loss of EGFR in those compartments, accumulation of DNA damage mediates tumor protein p53 (TP53)-dependent apoptosis. Second, EGFR controls hair layer–specific transcription factor expression to regulate epithelial hair layer specification and differentiation. EGFR deficiency leads to the absence of proper differentiation cues for hair shaft cells, resulting in loss of the medulla layer.

In a report of a patient treated with erlotinib who had pili torti, scalp biopsy study showed irregular thinning of the outer root sheath and disintegration of the inner root sheath.[8] Accordingly, perhaps changes in the outer and inner root sheaths might have caused the pili torti and the asymmetric hyperpigmented fusiform widening observed in our patients treated with EGFRI.

In addition to the well-documented cutaneous adverse effects of EGFRIs, including papulopustular rash, xerosis, and paronychia,[1,5] we observed in all 23 patients in this study a lesser known cutaneous adverse effect of seborrheic dermatitis–like changes macroscopically in the skin of the scalp, face, eyebrows, and eyelashes. This manifests on dermoscopy with white and yellow scale, whitish erythematous structureless areas, and mostly branching vessels (with a few dotted vessels), different from the classic dermoscopic features of seborrheic dermatitis which, similarly to other eczema subtypes, displays a patchy distribution of dotted vessels with yellow or white scales or crusts.[18,19] In contrast to the papulopustular rash, in our experience, dermatitis-like changes probably appear later in the course of treatment, in accordance with the macroscopic and trichoscopic hair changes.

The main limitation of this study is the lack of a trichoscopic-histologic correlation, which would have deepened our understanding of the pathology induced by iatrogenic inhibition of EGFR. The lack of a baseline assessment of the hair before EGFRI treatment is also a limitation, although all patients reported a clear and prominent change in the morphology and texture of their hair after treatment initiation. Furthermore, the absence of non-white patients is a limitation.

SUMMARY

In the present series, we describe trichoscopic shaft changes correlating to the known macroscopic hair changes in the scalp, eyebrows, and eyelashes induced by EGFRIs: the main trichoscopic changes were pili torti and asymmetric hyperpigmented fusiform widening of the hair shaft. All patients also had macroscopic seborrheic dermatitis–like changes in the skin of the scalp, eyebrows, and eyelashes, with dermoscopic manifestations not typical of seborrheic dermatitis. All findings were similar and consistent with all the EGFRIs, which points at a class affect and may contribute to our understanding of the role of EGFR in normal hair physiology.

Further magnification with light microscopy may provide a more accurate structure-based evaluation.

REFERENCES

1. Macdonald JB, Macdonald B, Golitz LE, LoRusso P, Sekulic A. Cutaneous adverse effects of targeted therapies: part I: inhibitors of the cellular membrane. *J Am Acad Dermatol*. 2015;72:203-218.
2. Holcmann M, Sibilia M. Mechanisms underlying skin disorders induced by EGFR inhibitors. *Mol Cell Oncol*. 2015;2:1-10.
3. Schneider MR, Werner S, Paus R, Wolf E. Beyond wavy hairs: the epidermal growth factor receptor and its ligands in skin biology and pathology. *Am J Pathol*. 2008;173:14-24.
4. Agero ALC, Dusza SW, Benvenuto-Andrade C, Busam KJ, Myskowski P, Halpern AC. Dermatologic side effects associated with the epidermal growth factor receptor inhibitors. *J Am Acad Dermatol*. 2006;55:657-670.
5. Lynch TJ, Kim ES, Eaby B, Garey J, West DP, Lacouture ME. Epidermal growth factor receptor inhibitor-associated cutaneous toxicities: an evolving paradigm in clinical management. *Oncologist*. 2007;12:610-621.
6. Fukui T, Kitamura H, Harada K, Nakano H, Sawamura D. Trichoscopic findings of erosive pustular dermatosis of the

scalp associated with gefitinib. *Case Rep Dermatol*. 2017;8562:44-49.
7. Amberg N, Sotiropoulou PA, Heller G, et al. EGFR controls hair shaft differentiation in a p53-independent manner. *iScience*. 2019;15:243-256.
8. Pirmez R, Piñeiro-Maceira J, Gonzalez C, Miteva M. Loose anchoring of anagen hairs and pili torti due to erlotinib. *Int J Trichology*. 2016;8:186-187.
9. Ena P, Fadda GM, Ena L, Farris A, Santeufemia DA. Tufted hair folliculitis in a woman treated with lapatinib for breast cancer. *Clin Exp Dermatol*. 2008;33:790-791.
10. Rosman IS, Anadkat MJ. Tufted hair folliculitis in a woman treated with trastuzumab. *Target Oncol*. 2010;5:295-296.
11. Rudnicka L, Rakowska A, Kerzeja M, Olszewska M. Hair shafts in trichoscopy: clues for diagnosis of hair and scalp diseases. *Dermatol Clin*. 2013;4:695-708.
12. Rudnicka L, Olszewska M, Waśkiel A, Rakowska A. Trichoscopy in hair shaft disorders. *Dermatol Clin*. 2018;4:421-430.
13. Penzi LR, Saavedra A, Senna MM. Long-standing pili torti in 2 patients with chronic graft-vs-host disease. *JAAD Case Rep*. 2018;4:44-46.
14. Mirmirani P, Samimi SS, Mostow E. Pili torti: clinical findings, associated disorders, and new insights into mechanisms of hair twisting. *Cutis*. 2009;84:143-147.
15. Raj G. Defects of the hair shaft. *DermNet NZ*. Available at: https://www.dermnetnz.org/topics/defects-of-the-hair-shaft/. Accessed October 22, 2019.
16. Gelles LN. Picture of the month. *Arch Pediatr Adolesc Med*. 1999;153:647.
17. Alexandrescu DT, Kauffman CL, Dasanu CA. The cutaneous epidermal growth factor network: can it be translated clinically to stimulate hair growth? *Dermatol Online J*. 2009;15(3):1.
18. Lallas A, Argenziano G, Apalla Z, et al. Dermoscopic patterns of common facial inflammatory skin diseases. *J Eur Acad Dermatol Venereol*. 2014;28(5):609-614.
19. Miteva M, Tosti A. Hair and scalp dermatoscopy. *J Am Acad Dermatol*. 2012;67(5):1040-1048.