

Frontal Fibrosing Alopecia Demographics: A Survey of 29 Patients

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PRACTICE POINTS

- Frontal fibrosing alopecia (FFA) may be associated with other autoimmune conditions, and patients should be screened accordingly.
- The most efficacious treatments for FFA include topical and intralesional steroids, hydroxychloroquine, calcineurin inhibitors, and excimer laser therapy.
- A stressful precipitating event or metal dental implants/fillings are 2 possible environmental triggers for this condition.

Frontal fibrosing alopecia (FFA) is a form of scarring alopecia whose diagnosis is increasing globally. Although its etiology is unknown, FFA is thought to be a clinical subset of lichen planopilaris (LPP) that primarily affects postmenopausal women. Patients diagnosed with FFA between January 2006 and December 2013 at clinics of the Washington University Division of Dermatology (St. Louis, Missouri) were studied using patient surveys and chart notes to assess demographics, clinical features, medical history, and treatment. Twenty-nine patients were enrolled in the study, including 28 women and 1 man. The average age of disease onset was 55.4 years (range, 29–75 years). Many patients (55%) had a history of autoimmune diseases, including hypothyroidism (35%), mucocutaneous lichen planus (28%), psoriasis (7%), vitiligo (3%), systemic lupus erythematosus (3%), iritis (3%), Sjögren syndrome (3%), and ulcerative colitis (3%). Patients often identified a stressful inciting event prior to onset of hair loss. Patients tried an average of 3 different treatments for hair loss, with topical and intralesional steroids, hydroxychloroquine, topical calcineurin inhibitors, and excimer laser therapy being the most efficacious at limiting hair loss.

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Frontal fibrosing alopecia (FFA) is a form of lymphocytic cicatricial alopecia that presents as frontotemporal hairline recession, typically in postmenopausal women.¹ The condition is considered to be a variant of lichen planopilaris (LPP) due to its similar histologic appearance.² Loss of eyebrow¹⁻¹¹ and body⁵⁻¹¹ hair also is commonly present in FFA, and histologic findings are identical to those for hair loss on the scalp,^{8,9} suggesting that FFA may be a form of generalized alopecia.

The pathogenesis of FFA is unknown, but several etiologies have been postulated. Some suggest that as a variant of LPP, FFA is a hair-specific autoimmune disorder characterized by a T cell-mediated immune reaction against epithelial hair follicle stem cells, leading to fibrosis and depletion of hair regeneration potential.¹² In support of this theory, FFA has been associated with other autoimmune diseases including hypothyroidism,^{6,8,13-16} mucocutaneous lichen planus,^{8,15,17} vitiligo,^{15,18} Sjögren syndrome,¹⁹ and lichen sclerosus et atrophicus.^{15,20} Another hypothesis suggests that the proandrogenic state in postmenopausal women may be related to the disease process.¹ This hypothesis is supported by the reported success of antiandrogen therapy with 5 α -reductase inhibitors (5 α -RIs) in stabilizing FFA.^{3-5,7} Finally, genetic^{16,21} and environmental factors related to smoking and socioeconomic status⁵ also have been postulated to be risk factors for FFA. A variety of treatments have shown varying success, including topical and intralesional corticosteroids, hydroxychloroquine, immunomodulators, antibiotics, and 5 α -RIs.^{1,3-6,8,15,17,22} However, FFA is considered to be relatively difficult to treat and commonly progresses regardless of treatment before spontaneously stabilizing.^{2-4,6,8,10}

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Since its discovery in 1994,¹ FFA has become increasingly prevalent, comprising 17% of new referrals for hair loss in one study (N=57).⁶ Although growing recognition of the condition likely plays a role in its increasing presentation, other unidentified factors may contribute to its expanding incidence. In this report, we describe the demographics, clinical features, and disease progression of 29 cases of FFA treated within our division using a series of surveys and chart reviews.

Methods

Upon receiving approval for the project from the institutional review board, we identified 29 patients who met the criteria for diagnosis of FFA through a chart review of all patients being treated for hair loss by clinics within the Washington University Division of Dermatology (St. Louis, Missouri). Diagnostic criteria for FFA included scarring alopecia in the frontotemporal distribution with associated perifollicular erythema or papules and, if performed, a scalp biopsy of the involved area of alopecia showing lymphocytic cicatricial alopecia, compatible with LPP. The diagnosis was confirmed by biopsy in 18 patients (62%), while the remainder of the diagnoses were made clinically. Most biopsy specimens were diagnosed by board-certified dermatopathologists at Washington University, with the remainder diagnosed by outside pathologists if the patient was initially diagnosed at another institution.

Patients meeting criteria for FFA were mailed a study consent form, as well as a 2-page survey to assess demographics, clinical features of hair loss, medical histories, social and family histories, and treatments utilized. After receiving consent from patients, survey results were collected and summarized. If there was any need for clarification of answers, follow-up questions were conducted via email prior to any data analysis that was performed.

For analysis of treatment response, patients were asked what treatments they had utilized and about the progression of their hair loss. Patients reporting stabilization of hair loss or hair regrowth were classified as treatment responsive. Patients who underwent multiple treatments were included in the analyses for each of those treatments. Physician records for treatment response were not correlated with patient responses due to inconsistent documentation, care received outside of our medical system, and prolonged or loss to follow-up. Physician-reported data were only used to identify qualifying patients and their biopsy results, as described above.

Results

Patient Demographics—Between October 2013 and May 2014, 29 patients with FFA were recruited into the study. Patients were diagnosed between January 2006 and December 2013. There were 28 female patients (97%) and 1 male patient (3%). The average age of disease onset was 55.4 years (range, 29–75 years). Twenty-five patients (86%) self-identified as non-Hispanic white,

3 patients (10%) as Asian, and 1 patient (3%) as black. Patients also appeared to be a more affluent group than the general St. Louis County population, with a median household income between \$75,000 and \$100,000. In comparison, the median household income reported in St. Louis County from 2008 to 2012 was \$58,485.²³ The patient population was primarily composed of nonsmokers, with 22 (76%) patients who had never smoked, 6 (21%) who were present smokers, and 1 (3%) smoked in the past. These results were comparable to the reported number of female smokers in Missouri.²⁴

Clinicopathologic Features—The clinical features of FFA are described in Table 1. All patients had frontotemporal recession of the hairline with some degree of scarring and perifollicular erythema (Figure 1). Most patients also reported hair loss at other sites, including 25 patients (86%) with eyebrow hair loss, 18 (62%) with limb hair loss, 11 (38%) with axillary hair loss, 11 (38%) with pubic hair loss, and 1 (3%) with eyelash hair loss. Patients also frequently reported inflammatory symptoms, including 19 patients (66%) with itching, 18 (62%) with redness, 3 (10%) with pain, 2 (7%) with papular lesions, and 1 (3%) with sores and erosions on the skin. Regarding progression of hair loss over time, 16 patients (55%) reported stabilization of hair loss, 11 (38%) reported progressive hair loss, and 2 (7%) reported some hair regrowth. Thirteen patients (45%) identified some inciting event that they believed to have triggered the disease. Ten patients (35%) identified stress as the inciting event, and 5 patients (17%) specifically referred to health-related stressors, including hip-replacement surgery, new diagnoses of systemic diseases, starting new medications, and stopping hormone replacement therapy. Furthermore, 2 (7%) patients reported exposure to chemicals and pesticides as suspected triggers.

Typical biopsy results showed a perifollicular lymphocytic infiltrate and fibrosis surrounding the infundibulum and isthmus of hair follicles (Figure 2). There were associated vacuolar changes in the basal layer and scattered dyskeratosis throughout the follicular epithelium. As



FIGURE 1. Frontal fibrosing alopecia presents with frontotemporal recession of the hairline with scarring and perifollicular erythema.

the disease progressed to end-stage scarring, there was marked reduction in the number of hair follicles, which were replaced by fibrous tracts, and a disappearance of the previous inflammatory infiltrate.

Medical History—Of the 26 female patients who provided data about menopause status at time of disease onset, 16 (62%) were postmenopausal, 5 (19%) were menopausal, and 5 (19%) were premenopausal. Of the 28 female patients in the study, 8 (29%) had a history of hysterectomy and 2 (7%) also had surgically induced menopause through bilateral surgical oophorectomy. Twenty-four patients (86%) had a childbearing history,

with an average of 2.3 children. Twelve patients (43%) reported use of hormone replacement therapy after menopause. Twelve patients (43%) also reported a history of oral contraceptive use.

Table 2 describes the comorbidities of all 29 patients. A history of autoimmune disease was prominent, found in 16 patients (55%). Thirteen patients (45%) reported thyroid disease, including 10 patients (35%) with hypothyroidism. Additionally, 8 patients (28%) had a history of mucocutaneous lichen planus, 2 (7%) of psoriasis/psoriatic arthritis,

TABLE 1. Clinical Features of Frontal Fibrosing Alopecia (N=29)

Clinical Feature	No. of Patients (%)
Nonscalp areas with hair loss	
None	3 (10)
Eyebrows	25 (86)
Arms	18 (62)
Legs	18 (62)
Axillae	11 (38)
Pubic hair	11 (38)
Eyelash	1 (3)
Symptoms	
None	7 (24)
Itching	19 (66)
Redness	18 (62)
Itching and redness	11 (38)
Pain	3 (10)
Papular lesions	2 (7)
Pain, itching, and redness	2 (7)
Triggers	
None	16 (55)
Stress	10 (35)
Other	3 (10)
Progression of symptoms	
Regrowth	2 (7)
Stable	16 (55)
Progressive	11 (38)

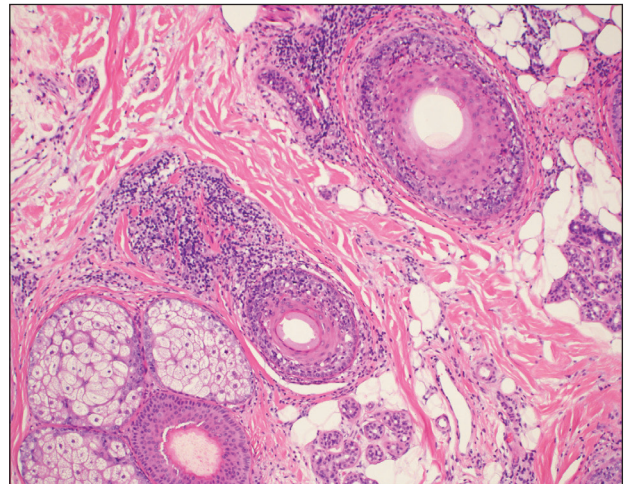


FIGURE 2. Frontal fibrosing alopecia demonstrates a moderately dense lymphocytic infiltrate surrounding the infundibula of hair follicles. Scattered necrotic keratinocytes also can be noted within the follicular epithelium (H&E, original magnification $\times 100$).

TABLE 2. Comorbidities in Patients With Frontal Fibrosing Alopecia (N=29)

Medical History	No. of Patients (%)
Thyroid disease	13 (45)
Hypothyroidism	10 (35)
Mucocutaneous lichen planus	8 (28)
Breast cancer	6 (21)
None	5 (17)
Psoriasis/psoriatic arthritis	2 (7)
Iritis	1 (3)
Sjögren syndrome	1 (3)
Systemic lupus erythematosus	1 (3)
Ulcerative colitis	1 (3)
Vitiligo	1 (3)

1 (3%) of vitiligo, 1 (3%) of systemic lupus erythematosus, 1 (3%) of iritis, 1 (3%) of Sjögren syndrome, and 1 (3%) of ulcerative colitis. Six patients (21%) also reported a history of breast cancer.

A dental history was obtained in 24 patients. All 24 patients reported having some dental implant or filling placed. Twenty-four patients (100%) had a history of metal amalgam implants, 8 (33%) had gold alloy implants, 4 (17%) had composite resin implants, and 3 (13%) had porcelain implants. Two patients had metal amalgam implants that had since been replaced by non-metal implants. Both patients reported no change in their clinical conditions with removal of the metal implants. Six of 8 patients (75%) with mucocutaneous lichen planus reported having dental implants. Of them, all 6 patients (100%) reported having metal amalgam implants, and 3 patients (50%) additionally reported having gold alloy implants.

Treatments—On average, patients were treated with 3 different therapies for FFA (range, 0–14). The treatments utilized are listed in Table 3, and responses to treatments are summarized in Table 4. Topical steroids were the most popular treatment modality and were used by 21 patients (72%). Approximately half of those patients reported treatment response with stabilization of hair loss or regrowth (n=11; 52%). Hydroxychloroquine was the second most commonly used modality (16 patients [55%]), with 10 of those patients (63%) reporting treatment response. Intralesional steroids were used in 11 patients (38%), with a treatment response in 36% (4/11) of those patients. Topical pimecrolimus and tacrolimus were used by 6 patients (21%), with 5 of those patients (83%) reporting treatment response. UVB excimer laser therapy was used on 3 patients (10%) with 100% treatment response.

Treatments with little or no treatment response to hair loss include doxycycline, minocycline, and topical minoxidil. Seven patients (24%) were treated with doxycycline or minocycline, all of whom reported no clinical response. Topical minoxidil was used by 3 patients (10%), with only 1 patient (33%) reporting stabilization of hair loss but no regrowth of hair. 5 α -reductase inhibitors such as finasteride and dutasteride were only used by 1 patient (3%), who reported no treatment response. Other treatments that were rarely used include meloxicam (n=2), azathioprine (n=2), oral clindamycin (n=2), bimatoprost (n=1), quinacrine (n=1), cephalosporin (n=1), prednisone (n=1), isotretinoin (n=1), methotrexate (n=1), spironolactone (n=1), topical clindamycin (n=1), and laser hair removal (n=1). Of these, only meloxicam and quinacrine were anecdotally associated with stabilization of hair loss, while the rest of the treatments were associated with progressive hair loss despite therapy.

Comment

Frontal fibrosing alopecia is a form of cicatricial alopecia considered to be a clinical subset of LPP. Although the pathogenesis of both diseases are poorly understood,

LPP is the better-studied model and is generally considered to be an autoimmune disease specific to the hair follicle, involving a cell-mediated inflammatory response to epithelial hair follicle stem cells.¹² In support of this hypothesis, FFA and LPP have been frequently associated with autoimmune diseases, particularly with hypothyroidism.^{6,13-15} We found that 55% of our patients had a history of autoimmune disease, including 35% with hypothyroidism, 28% with mucocutaneous lichen planus, 7% with psoriasis, 3% with vitiligo, 3% with systemic lupus erythematosus, 3% with iritis, 3% with Sjögren syndrome, and 3% with ulcerative colitis. The link between FFA and hypothyroidism has

TABLE 3. Frontal Fibrosing Alopecia Treatments (N=29)^a

Treatment	No. of Patients (%)
Topical steroids	21 (72)
Hydroxychloroquine	16 (55)
Intralesional steroids	11 (38)
Topical pimecrolimus	5 (17)
Doxycycline	4 (14)
Minocycline	3 (10)
Topical minoxidil	3 (10)
UVB excimer laser	3 (10)
Azathioprine	2 (7)
Clindamycin	2 (7)
Meloxicam	2 (7)
Bimatoprost	1 (3)
Cephalosporin	1 (3)
Dutasteride	1 (3)
Finasteride	1 (3)
Isotretinoin	1 (3)
Laser hair removal	1 (3)
Methotrexate	1 (3)
Prednisone	1 (3)
Quinacrine	1 (3)
Spirolactone	1 (3)
Topical clindamycin	1 (3)
Topical tacrolimus	1 (3)

^aThe mean number of treatments was 3 (range, 0–14).

TABLE 4. Frontal Fibrosing Alopecia Treatment Response^a

Treatment	No. of Treatments	Clinical Progression, n (%)		
		Regrowth	Stable	Progressive
Topical steroids	21	1 (5)	10 (48)	10 (48)
Hydroxychloroquine	16	1 (6)	9 (56)	6 (38)
Intralesional steroids	11	1 (9)	3 (27)	7 (64)
Topical pimecrolimus	5	1 (20)	4 (80)	0 (0)
Doxycycline	4	0 (0)	0 (0)	4 (100)
Minocycline	3	0 (0)	0 (0)	3 (100)
UVB excimer laser	3	1 (33)	2 (67)	0 (0)
Topical minoxidil	3	0 (0)	1 (33)	2 (67)

^aTreatment response only listed for most frequently used treatments.

been the best studied, with a large study by Atanskova Mesinkovska et al¹⁴ finding that 34% of 166 patients with LPP and FFA have some kind of thyroid disease and 29% have hypothyroidism. Frontal fibrosing alopecia also has been associated with mucocutaneous lichen planus,^{8,15,17} vitiligo,^{15,18} Sjögren syndrome,¹⁹ and lichen sclerosus et atrophicus^{15,19} in other studies, but the current study also identifies less frequently reported associations between FFA and psoriasis, iritis, and ulcerative colitis.

Although FFA has been classically described to affect postmenopausal women, recent studies have consistently identified that premenopausal women^{4-6,8,16,17} and men^{14,16} also can be affected by the condition. In our patient cohort, there was 1 male patient (3%), and a substantial number of the female patients were premenopausal (19%) and menopausal (19%) at the time of disease onset. Most of the patients studied were white; Asian and black patients were a consistent minority across FFA studies,^{5,13-16,25} highlighting the importance of screening for FFA in all demographics.

In our study, FFA patients also appeared to be more affluent than the general population and were predominantly nonsmokers (76%). These statistics are consistent with the United Kingdom population studied by MacDonald et al,⁶ which demonstrated a higher socioeconomic status and higher incidence of nonsmoking in their cases of FFA. Another large retrospective study of FFA patients in Spain found that 87% of their FFA cases (N=355) were nonsmokers, though they did not note a difference from the general unaffected population.¹⁵ In our study, we replicated these trends, finding an above average affluence level and a high but not statistically significant incidence of nonsmokers. Although it is not clear how socioeconomic status or smoking factors into

the pathology of FFA, these studies may show a general trend in the environmental demographics of the disease.

Clinically, patients with FFA typically present with hair loss of the scalp as well as other sites. The eyebrows are the most common site to be affected outside of the scalp, affecting 86% of our patients, whereas eyelashes are the least commonly affected, presenting in only 3% of our patients. Body hair loss also is common, with almost two-thirds of our cohort reporting hair loss on the limbs and more than one-third reporting loss of axillary and pubic hair. These findings are consistent with those of other studies.^{3-6,8,13,15} Eyelash loss, body hair loss, and facial papules have been found to be associated with more severe forms of FFA,¹⁵ though we did not investigate these forms in our study. Inflammatory symptoms are common, with pruritus affecting 66% of our patients and pain affecting 10% of patients, consistent with the published literature.^{3,13,15,17}

Multiple studies have shown that female FFA patients have a higher incidence of hysterectomies in their medical history.^{5,8,15} This observation has been used to further support the hypothesis that a change in sex hormone balance may trigger the initial onset of disease.^{5,8,15} A considerable number of the female patients in our study had also undergone hysterectomies (29%). Only 2 patients (7%) underwent premature surgical menopause through bilateral removal of the ovaries, and neither of these patients had abnormally early onset of FFA (age at onset, 52 and 65 years). Many patients in our study also reported a history of pharmacologic manipulation of sex hormones with hormone replacement therapy (43%) and oral contraceptive use (43%). However, patients with FFA have not been identified to have abnormal hormone levels compared to unaffected postmenopausal

women.¹ Additionally, the disease does not exclusively affect androgen-dependent hair, as indicated by the high prevalence of eyebrow hair loss. We hypothesize that the link between increased prevalence of hysterectomy and FFA is not due to hormonal changes but rather from the stresses related to the hysterectomy or associated conditions that required the surgery. In our study, 35% of patients identified stress as the inciting event prior to their onset of hair loss, with 17% specifically referring to health-related stress such as surgery or new diagnoses as the cause. Although this pattern is purely observational, it is valuable to consider that stress could contribute to the initial onset of FFA as with alopecia areata.²⁶

A dental history was obtained in 24 patients to explore the possibility of FFA as a manifestation of contact allergy secondary to exposure to metal dental implants. Contact allergies to metal amalgam and gold alloy dental implants/fillings frequently have been described as presenting as oral lichen planus in the literature.²⁷⁻³⁴ Given the histologic overlap between oral lichen planus and LPP/FFA, it is worth exploring the possibility that LPP and FFA are other manifestations of contact allergic response. In our study, 100% of the patients who provided a dental history had metal amalgam implants and 33% had gold alloy implants. It is an interesting observation, but it should be noted that none of the patients in our study had undergone patch testing for contact allergies to the metals in their dental implants, and further studies are required to explore this hypothesis.

Frontal fibrosing alopecia is a difficult condition to treat. In our study, patients tried an average of 3 different treatments, the most common being topical steroids (72%), hydroxychloroquine (55%), and intralesional steroids (38%). 5 α -reductase isozymes were rarely utilized for patients in this study. Treatment response was noted in most patients using topical steroids, hydroxychloroquine, topical calcineurin inhibitors, and excimer laser therapy. Intralesional steroids also were efficacious in about 36% of patients treated. Little to no treatment response was reported in patients using doxycycline, minocycline, and topical minoxidil.

A PubMed search of articles indexed for MEDLINE using the terms *randomized control trial* and *frontal fibrosing alopecia* yielded no randomized controlled trials that have been performed to demonstrate the most efficacious treatments of FFA. However, one systematic review of 114 patients found 5 α -RIs, antimalarials, and intralesional corticosteroids to yield the best responses in treating FFA.²² Another large, multicenter, retrospective study of 355 patients also demonstrated that 5 α -RIs and intralesional corticosteroids minimized hair loss most effectively across treatment modalities.¹⁵ One treatment that was not discussed in either study but was utilized in ours was the UVB excimer laser, which has been demonstrated to induce T-cell apoptosis and decrease inflammation in psoriasis but has been infrequently studied in the use of FFA or LPP. In one study of 13 patients with

LPP, excimer laser treatment was successful in reducing inflammatory symptoms and improving hair loss.³⁵ Our results reaffirm that laser therapy could be considered more frequently as a treatment of FFA.

This study is subject to several limitations. The study size was comprised of a relatively small number of patients with the condition. Additionally, only one-third of patients contacted agreed to participate in the study, and therefore the responses received may not be completely representative of all FFA patients. With a retrospective study, there is potential for recall bias in the data that are collected. Physician chart correlation to patient responses could not be reliably performed due to inconsistent documentation, care received outside our medical system, and prolonged or loss to follow-up. Another concern is that not all diagnoses of FFA in this study were biopsy confirmed. In one patient with systemic lupus erythematosus who declined biopsy, it cannot be confirmed that her etiology of scarring alopecia was FFA rather than discoid lupus erythematosus. Finally, because patients were treated with multiple medications, often concurrently, it was difficult to parse out which medications were efficacious and which were not. Despite these limitations, the findings in the study add to the growing literature about a rare but increasingly prevalent presentation.

Conclusion

Frontal fibrosing alopecia is a condition that predominantly affects white postmenopausal women but should not be overlooked in other demographics; higher socioeconomic status and nonsmoking are consistent with cases of FFA worldwide. Alopecia frequently involves other body hair, particularly the eyebrows, and is commonly associated with pruritus and pain. Many patients can identify an inciting event, usually stress, a health crisis, or new external exposures that they believe to have triggered the event. Consistent with data about LPP, FFA is frequently associated with autoimmune conditions, particularly hypothyroidism. A substantial portion of patients with FFA have had metal amalgam or gold alloy dental implants placed, though no patch testing was done to confirm that these patients have a contact allergy to these metals. Treatment for the condition is difficult, but topical and intralesional steroids, hydroxychloroquine, calcineurin inhibitors, and excimer laser therapy are efficacious in a large proportion of patients. Nevertheless, further research through prospective randomized trials is necessary to determine the best treatment modalities for FFA. Frontal fibrosing alopecia is a scarring form of hair loss that causes substantial emotional distress; therefore, it is critical to continue to investigate its etiology and treatments to improve patient care.

REFERENCES

1. Kossard S. Postmenopausal frontal fibrosing alopecia: scarring alopecia in a pattern distribution. *Arch Dermatol.* 1994;130:770-774.
2. Kossard S, Lee MS, Wilkinson B. Postmenopausal frontal fibrosing alopecia: a frontal variant of lichen planopilaris. *J Am Acad Dermatol.* 1997;36:59-66.

3. Tosti A, Piraccini BM, Iorizzo M, et al. Frontal fibrosing alopecia in postmenopausal women. *J Am Acad Dermatol*. 2005;52:55-60.
4. Moreno-Ramírez D, Camacho Martínez F. Frontal fibrosing alopecia: a survey in 16 patients. *J Eur Acad Dermatol Venereol*. 2005;19:700-705.
5. Ladizinski B, Bazakas A, Selim MA, et al. Frontal fibrosing alopecia: a retrospective review of 19 patients seen at Duke University. *J Am Acad Dermatol*. 2013;68:749-755.
6. MacDonald A, Clark C, Holmes S. Frontal fibrosing alopecia: a review of 60 cases. *J Am Acad Dermatol*. 2012;67:955-961.
7. Georgala S, Katoulis AC, Befon A, et al. Treatment of postmenopausal frontal fibrosing alopecia with oral dutasteride. *J Am Acad Dermatol*. 2009;61:157-158.
8. Tan KT, Messenger AG. Frontal fibrosing alopecia: clinical presentations and prognosis. *Br J Dermatol*. 2009;160:75-79.
9. Chew AL, Bashir SJ, Wain EM, et al. Expanding the spectrum of frontal fibrosing alopecia: a unifying concept. *J Am Acad Dermatol*. 2010;63:653-660.
10. Miteva M, Camacho I, Romanelli P, et al. Acute hair loss on the limbs in frontal fibrosing alopecia: a clinicopathological study of two cases. *Br J Dermatol*. 2010;163:426-428.
11. Abbas O, Chedraoui A, Ghosn S. Frontal fibrosing alopecia presenting with components of Piccardi-Lassueur-Graham-Little syndrome. *J Am Acad Dermatol*. 2007;57(2 suppl):S15-S18.
12. Harries MJ, Meyer K, Chaudhry I, et al. Lichen planopilaris is characterized by immune privilege collapse of the hair follicle's epithelial stem cell niche. *J Pathol*. 2013;231:236-247.
13. Dlova NC, Jordaan HF, Skenjane A, et al. Frontal fibrosing alopecia: a clinical review of 20 black patients from South Africa. *Br J Dermatol*. 2013;169:939-941.
14. Atanaskova Mesinkovska N, Brankov N, Piliang M, et al. Association of lichen planopilaris with thyroid disease: a retrospective case-control study. *J Am Acad Dermatol*. 2014;70:889-892.
15. Vañó-Galván S, Molina-Ruiz AM, Serrano-Falcón C, et al. Frontal fibrosing alopecia: a multicenter review of 355 patients. *J Am Acad Dermatol*. 2014;70:670-678.
16. Dlova N, Goh CL, Tosti A. Familial frontal fibrosing alopecia. *Br J Dermatol*. 2013;168:220-222.
17. Samrao A, Chew AL, Price V. Frontal fibrosing alopecia: a clinical review of 36 patients. *Br J Dermatol*. 2010;163:1296-1300.
18. Miteva M, Aber C, Torres F, et al. Frontal fibrosing alopecia occurring on scalp vitiligo: report of four cases. *Br J Dermatol*. 2011;165:445-447.
19. Sato M, Saga K, Takahashi H. Postmenopausal frontal fibrosing alopecia in a Japanese woman with Sjögren's syndrome. *J Dermatol*. 2008;35:729-731.
20. Feldmann R, Harms M, Saurat JH. Postmenopausal frontal fibrosing alopecia. *Hautarzt*. 1996;47:533-536.
21. Junqueira Ribeiro Pereira AF, Vincenzi C, Tosti A. Frontal fibrosing alopecia in two sisters. *Br J Dermatol*. 2010;162:1154-1155.
22. Rácz E, Gho C, Moorman PW, et al. Treatment of frontal fibrosing alopecia and lichen planopilaris: a systematic review. *J Eur Acad Dermatol Venereol*. 2013;27:1461-1470.
23. QuickFacts: St. Louis County, Missouri. United States Census Bureau website. <https://www.census.gov/quickfacts/fact/table/stlouiscountymissouri/PST045217>. Accessed February 6, 2019.
24. State tobacco activities tracking and evaluation (STATE) system. State highlights. Centers for Disease Control and Prevention website. https://nccd.cdc.gov/STATESystem/rdPage.aspx?rdReport=OSH_STATE.Highlights. Accessed February 6, 2019.
25. Miteva M, Whiting D, Harries M, et al. Frontal fibrosing alopecia in black patients. *Br J Dermatol*. 2012;167:208-210.
26. Willemssen R, Vanderlinden J, Roseeuw D, et al. Increased history of childhood and lifetime traumatic events among adults with alopecia areata. *J Am Acad Dermatol*. 2009;60:388-393.
27. Segura-Egea JJ, Bullón-Fernández P. Lichenoid reaction associated to amalgam restoration. *Med Oral Patol Oral Cir Bucal*. 2004;9:421-424.
28. Laeijendecker R, van Joost T. Oral manifestations of gold allergy. *J Am Acad Dermatol*. 1994;30:205-209.
29. Marcusson JA. Contact allergies to nickel sulfate, gold sodium thiosulfate and palladium chloride in patients claiming side-effects from dental alloy components. *Contact Dermatitis*. 1996;34:320-323.
30. Nordlind K, Lidén S. Patch test reactions to metal salts in patients with oral mucosal lesions associated with amalgam restorations. *Contact Dermatitis*. 1992;27:157-160.
31. Koch P, Bahmer FA. Oral lichenoid lesions, mercury hypersensitivity and combined hypersensitivity to mercury and other metals: histologically-proven reproduction of the reaction by patch testing with metal salts. *Contact Dermatitis*. 1995;33:323-328.
32. Laine J, Kalimo K, Happonen RP. Contact allergy to dental restorative materials in patients with oral lichenoid lesions. *Contact Dermatitis*. 1997;36:141-146.
33. Yiannias JA, el-Azhary RA, Hand JH, et al. Relevant contact sensitivities in patients with the diagnosis of oral lichen planus. *J Am Acad Dermatol*. 2000;42:177-182.
34. Scalf LA, Fowler JF Jr, Morgan KW, et al. Dental metal allergy in patients with oral, cutaneous, and genital lichenoid reactions. *Am J Contact Dermat*. 2001;12:146-150.
35. Navarini AA, Kolios AG, Prinz-Vavricka BM, et al. Low-dose excimer 308-nm laser for treatment of lichen planopilaris. *Arch Dermatol*. 2011;147:1325-1326.