To prepare this article, we reviewed the existing literature on frontal fibrosing alopecia. We searched MEDLINE and EMBASE using the term “frontal fibrosing alopecia” and, by selecting descriptive studies with 4 or more patients, we identified a total of 7 studies that included 78 patients with frontal fibrosing alopecia (Table 1). The isolated case studies were rejected due to the heterogeneous nature of the data provided. No other types of primary studies or clinical trials on the treatment of patients with frontal fibrosing alopecia were found. All the available clinical, pathological, and therapeutic information was extracted from the same published descriptive series and case studies.

Diagnostic Assessment of Frontal Fibrosing Alopecia

Frontal fibrosing alopecia is a disease that is diagnosed clinically in most cases and for which a histopathological study is indicated in patients for whom the clinical diagnosis...
is not conclusive, taking into consideration a number of recommendations that will be described below. Further tests have not provided any benefit in the diagnosis of frontal fibrosing alopecia.

**Clinical Diagnosis**

Frontal fibrosing alopecia is characterized by the recession of the frontal hairline with scarring of the alopecic skin, often accompanied by alopecia of the eyebrows, and usually occurs in postmenopausal women1, 2 (Figure 1). It is therefore a clinically well-differentiated entity. However, with the exception of the receding hairline present in 100% of patients, the other clinical manifestations reported for frontal fibrosing alopecia appear with variable frequency and are therefore of varying interest in clinical diagnosis (Table 2).

Scarring of the alopecic skin, onset after menopause, presence of perifollicular papules, and eyebrow alopecia appeared in more than 60% of patients with frontal fibrosing alopecia in the clinical series reviewed (Table 2). Other manifestations, such as follicular hyperkeratosis, associated female androgenetic alopecia, axillary alopecia, pruritus, and lichen planus or lichen planopilaris, were identified in less than 30% patients with frontal fibrosing alopecia (Table 2). Following are the clinical manifestations reported in patients with frontal fibrosing alopecia:

**Recession of the Frontal Hairline**

The progressive recession of the frontal and parietal hairline is the most constant and characteristic clinical manifestation of frontal fibrosing alopecia. It is present in all patients and is therefore a requisite condition for diagnosis. Onset of

---

**Table 1. Case Series of Patients With Frontal Fibrosing Alopecia**

<table>
<thead>
<tr>
<th>Author</th>
<th>No.</th>
<th>Study Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kossard1 1994</td>
<td>6</td>
<td>Clinical, pathologic, therapeutic</td>
</tr>
<tr>
<td>Kossard et al2 1997</td>
<td>10</td>
<td>Clinical, pathologic, therapeutic</td>
</tr>
<tr>
<td>Naz et al3 2003</td>
<td>4</td>
<td>Clinical, therapeutic</td>
</tr>
<tr>
<td>Vaisse et al4 2003</td>
<td>20</td>
<td>Clinical, pathologic, therapeutic</td>
</tr>
<tr>
<td>Moreno-Ramírez et al5 2005</td>
<td>16</td>
<td>Clinical, pathologic, therapeutic</td>
</tr>
<tr>
<td>Tosti et al6 2005</td>
<td>14</td>
<td>Clinical, therapeutic</td>
</tr>
<tr>
<td>Poblet et al7 2006</td>
<td>8</td>
<td>Clinical, pathologic</td>
</tr>
<tr>
<td>Total</td>
<td>78</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 1.** Receding frontal hairline in a 58-year-old woman. Complete loss of eyebrow hair can be seen that has been covered up by the patient using makeup.

**Table 2. Clinical Manifestations of Frontal Fibrosing Alopecia (n = 78)**

<table>
<thead>
<tr>
<th>Clinical Manifestation</th>
<th>No.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal recession</td>
<td>78</td>
<td>100%</td>
</tr>
<tr>
<td>Scarring alopecia</td>
<td>75</td>
<td>96.15%</td>
</tr>
<tr>
<td>Postmenopausal onset</td>
<td>74</td>
<td>94.87%</td>
</tr>
<tr>
<td>Perifollicular papules</td>
<td>49b</td>
<td>72.06%</td>
</tr>
<tr>
<td>Eyebrow alopecia</td>
<td>49</td>
<td>62.82%</td>
</tr>
<tr>
<td>Follicular hyperkeratosis</td>
<td>24</td>
<td>30.77%</td>
</tr>
<tr>
<td>Female androgenetic alopecia</td>
<td>16</td>
<td>20.51%</td>
</tr>
<tr>
<td>Axillary alopecia</td>
<td>11</td>
<td>14.10%</td>
</tr>
<tr>
<td>Pruritus</td>
<td>7</td>
<td>8.97%</td>
</tr>
<tr>
<td>Premenopausal onset</td>
<td>4</td>
<td>5.13%</td>
</tr>
<tr>
<td>Lichen planus</td>
<td>4</td>
<td>5.13%</td>
</tr>
</tbody>
</table>

*aResult of the review of case studies described in the text. Clinical manifestations that appear in more than 60% of cases are marked in bold typeface. The mean age of the patients was 63.5 years.

bFrom 68 patients.
recession of the occipital hairline was also included as frontal fibrosing alopecia.  

**Scarring Alopecia**

Changes in the alopecic area consisting of pale skin with destruction of the follicular openings and skin atrophy are described in 96% of published cases of patients with frontal fibrosing alopecia. This scarring is usually mild and no induration or clinically evident sclerosis has been reported. The affected scalp area has an unusual appearance that is clearly different from hyperpigmentation of the forehead due to chronic sun damage. These changes are common to other entities associated with scarring alopecia and are therefore not useful for the differential diagnosis.

**Onset in Postmenopausal Women**

The slow progression of the disease and the mildness of the initial stages make it difficult to estimate the exact age at which onset of the disease occurs, with delays in diagnosis of between 1 and 18 years.  

In the published studies, the age at onset of frontal fibrosing alopecia varied between 45 and 82 years of age, with a mean age for all reviewed cases of 63.15 years.

Frontal fibrosing alopecia was initially described in postmenopausal women and this led to coining the term “postmenopausal frontal fibrosing alopecia.” Onset after the menopause is reported in 94.87% of patients in the published studies. Therefore, onset of frontal fibrosing alopecia occurred in premenopausal women in 5.13% of cases. Despite the fact that onset is postmenopausal in a significant majority of cases and that hormone replacement therapy has no effect on the course of the alopecia, some authors prefer to dissociate the term frontal fibrosing alopecia from any reference to the hormonal status of the patient.

**Erythema, Papules, and Perifollicular Inflammation**

Inflammatory papules and follicular or perifollicular erythema in the line of progression of the alopecia have been reported in 72% of patients with frontal fibrosing alopecia (Figure 3). These manifestations, which are usual in lichen planopilaris, are present particularly in the initial stages of frontal fibrosing alopecia and correlate with the inflammatory phase of the disease. The presence of these lesions, together with the usual signs, led to frontal fibrosing alopecia.
alopecia being considered a clinical subtype of lichen planopilaris.³

Eyebrow Alopecia

The thinning or partial or total loss of eyebrow hair has been reported in 62.82% of patients with frontal fibrosing alopecia. Hair-loss from the lateral third of the eyebrows is characteristic and, in some cases, there may be total loss of eyebrow hair (Figures 2 and 3A). In other cases, however, diffuse thinning of the eyebrows occurs, giving them a sparse appearance. Eyelash loss was observed in a patient in the series published by Kossard et al.² Eyebrow alopecia is also a frequent characteristic of alopecia areata; several patients with frontal fibrosing alopecia from the reviewed studies were initially diagnosed with alopecia areata due to the total or partial loss of eyebrow hair.

Follicular Hyperkeratosis

Follicular keratotic plugs or hyperkeratotic papules have been reported in 30.77% of patients with frontal fibrosing alopecia and should be distinguished from the lesions that occur in widespread form on the trunk and limbs of patients with Graham–Little–Piccardi–Lassueur syndrome.

Female Androgenetic Alopecia

Of the patients with frontal fibrosing alopecia included in the reviewed series, 20% presented different degrees of female pattern baldness. This association, which is a consequence of the age of the patient with frontal fibrosing alopecia, gives rise to a clinical pattern of recession of the frontal hairline and diffuse alopecia on the rest of the scalp.⁵

Axillary Alopecia

Generalized thinning of the hair on other parts of the body, particularly the axillae, was reported in 14.10% of the cases from the reviewed series. This loss of axillary hair may be accompanied by reduced hair density in other areas (pubic area, limbs etc) and this may, in some cases, be accompanied by mild skin atrophy and diffuse follicular erythema.² In some cases, this loss of hair from the axillae and limbs was interpreted as being compatible with Piccardi–Lassueur–Graham–Little syndrome, especially in cases with follicular keratotic papules—a finding present in all patients with this syndrome. However, recession of the frontal hairline points towards frontal fibrosing alopecia rather than a diagnosis of multifocal scarring alopecia.²

Pruritus

Pruritus of the scalp was reported by 8% of patients with frontal fibrosing alopecia.

Associated Dermatoses

Unlike lichen planopilaris, which is associated with lichen planus lesions in up to 50% of patients, only 5% of patients with frontal fibrosing alopecia presented lesions compatible with lichen planus in other locations. Some patients with frontal fibrosing alopecia have tested positive for antinuclear antibodies, although patients with lupus erythematosus² or other autoimmune diseases were not described in the reviewed studies.

Histopathological Diagnosis

The histopathological signs originally described by Kossard¹,² and confirmed in subsequent studies consisted of the presence of a lichenoid perifollicular infiltrate at the level of the isthmus and infundibulum, with perifollicular fibrosis in onion–skin layers and lamellar fibrosis at the same location as the inflammatory infiltrate. These histopathological manifestations—shown in the 16 patients of the series of Kossard et al.²—were indistinguishable from the histopathological changes in the cases of multifocal lichen planopilaris. However, in the clinical series reported by Poblet et al.⁷ dermatological abnormalities were described that may allow for a differential diagnosis between the 2 entities (Figure 4 and Table 3).
Therefore, in cases where the clinical manifestations are not sufficient to diagnose frontal fibrosing alopecia, the following recommendations should be followed to provide an adequate histopathological study:

1. Perform 6-8 mm punch biopsies in the area of progression of the alopecia where hair still exists, including follicles and, if evident, perifollicular papules.
2. Transverse sections should be prepared to facilitate observation of the infiltrate and perifollicular fibrosis.
3. It should be remembered that biopsies in patients with long-term frontal fibrosing alopecia will present fibrous tracts with no follicles and no inflammatory infiltrate. These biopsies should therefore be reported as “scarring alopecia,” which, from a pathological point of view, is indistinguishable from other diseases.

### Additional Tests

Several further tests were performed in the published studies, including a complete blood count and general biochemistry, liver function tests, thyroid hormones (thyrotropin, triiodothyronine, thyroxine), serology for hepatitis C virus, antinuclear antibodies, sex hormones (luteinizing hormone, follicle stimulating hormone, androgens, and estradiol), and prolactin. Results were normal in most patients, with isolated cases of positive results for antinuclear antibodies at low titers.

Therefore, based on the results of the reviewed studies, further tests are not required in patients with clinical symptoms compatible with frontal fibrosing alopecia who present no other dermatosis or associated systemic disease and no other clinical manifestations of hyperandrogenism.

### Differential Diagnosis

Frontal fibrosing alopecia is a form of primary scarring alopecia. This is a large group of diseases characterized by the irreversible loss of hair due to a scarring process that leads to the thinning and destruction of the hair follicles—generally in the scalp. The distribution pattern of the alopecia (single plaque, multifocal, hairline recession), the involvement of other hairy areas (eyebrows, axillae, body hair), and the presence of other associated cutaneous manifestations (signs of lichen planus, discoid lupus, etc) will allow for a clinical differential diagnosis with these diseases in a large number of cases. Histopathological findings may be of help, especially in the initial stages where each of the diseases being considered will show a more or less typical inflammatory infiltrate, whereas the final stages of scarring alopecia are characterized by the presence of fibrosis of the follicle and absence of inflammatory infiltrate.

Frontal fibrosing alopecia should also be differentiated from other forms of nonscarring alopecia that may have similar clinical symptoms and course (progressive recession of the frontal hairline).

Table 4 shows the clinical characteristics of diseases that may pose problems in the differential diagnosis with frontal fibrosing alopecia.

### Treatment

The fact that frontal fibrosing alopecia presents the symptoms of scarring alopecia in the final stages and that progression is slow, with spontaneous cessation of the disease years after onset makes both treating the disease...
and assessing the effectiveness of the administered treatment difficult.

The available evidence on the treatment of frontal fibrosing alopecia comes from retrospective observational studies that described the clinical manifestations of the disease. There are no randomized clinical trials or quasi-experimental studies that make it possible to reach conclusions regarding the most appropriate treatment options. Table 5 describes the treatment regimens and combinations used, along with the results obtained. Each of these alternatives was tried in less than 10 patients, making the level of evidence insufficient to establish recommendations for each of the options described below.

### Corticosteroids

Corticosteroids may be considered a rational approach in the initial stages of the disease, characterized by the presence of perifollicular papules and lymphocytic infiltrate. The relationship between frontal fibrosing alopecia and lichen planopilaris and lichen planus would also support the use of corticosteroids in these patients. Of the patients

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**Table 4. Differential Diagnosis of Frontal Fibrosing Alopecia**

<table>
<thead>
<tr>
<th>Scarring alopecia</th>
<th>Age/sex</th>
<th>Pattern of scalp alopecia</th>
<th>Scarring alopecia atrophy, follicular destruction</th>
<th>Perifollicular erythema/inflammation</th>
<th>Eyebrow alopecia</th>
<th>Axillary alopecia</th>
<th>Other key diagnostic signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal alopecia</td>
<td>60-year-old woman</td>
<td>Receding frontal hairline</td>
<td>Yes</td>
<td>+</td>
<td>Yes</td>
<td>Yes</td>
<td>See text</td>
</tr>
<tr>
<td>Discoid lupus</td>
<td>Young woman</td>
<td>Multifocal plaques</td>
<td>Yes</td>
<td>++</td>
<td>No</td>
<td>No</td>
<td>ANA positive in 50% Other manifestations of discoid lupus</td>
</tr>
<tr>
<td>Lichen planopilaris</td>
<td>Middle-aged woman</td>
<td>Multifocal plaques</td>
<td>Yes</td>
<td>++</td>
<td>No</td>
<td>No</td>
<td>See text</td>
</tr>
<tr>
<td>Traction alopecia</td>
<td>Receding frontal hairline</td>
<td>Yes</td>
<td>–</td>
<td>No</td>
<td>No</td>
<td>Continuous traction due to tied-back hair Irregular edges and broken hairs</td>
<td></td>
</tr>
<tr>
<td>Pseudopelade</td>
<td>40-year-old woman</td>
<td>Multifocal plaque</td>
<td>Yes</td>
<td>+ Initial stages</td>
<td>No</td>
<td>No</td>
<td>–</td>
</tr>
<tr>
<td>Graham-Little-Piccardi-Lassueur syndrome</td>
<td>30-70-year-old woman</td>
<td>Multifocal plaques</td>
<td>Yes</td>
<td>++</td>
<td>Yes</td>
<td>Yes</td>
<td>Follicular keratotic papules on trunk and limbs</td>
</tr>
<tr>
<td>Alopecia areata</td>
<td>Peak in second and fourth decades of life</td>
<td>Multifocal plaque</td>
<td>No</td>
<td>–</td>
<td>Yes</td>
<td>No</td>
<td>Peribulbar lymphocytic infiltrate</td>
</tr>
<tr>
<td>Female pattern balding</td>
<td>Middle-aged/elderly woman</td>
<td>Diffuse alopecia with respect to the frontal hairline</td>
<td>No</td>
<td>–</td>
<td>No</td>
<td>No</td>
<td>Follicular miniaturization</td>
</tr>
<tr>
<td>High family hairline</td>
<td>Receding frontal hairline</td>
<td>No</td>
<td>–</td>
<td>No</td>
<td>No</td>
<td>–</td>
<td>Miniaturization or absence of follicles, with no inflammation</td>
</tr>
</tbody>
</table>

Abbreviation: ANA, antinuclear antibodies.
Table 5. Treatment Regimens Administered to Patients With Frontal Fibrosing Alopecia

<table>
<thead>
<tr>
<th>Study</th>
<th>Therapeutic Regimen</th>
<th>No.</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kossard1 1994 and Kossard et al 1997</td>
<td>Oral prednisone 25-50 mg/d for 1 month</td>
<td>4</td>
<td>2 cases of temporary slowing of hair loss (50%) 2 cases with no response and slow progression (50%)</td>
</tr>
<tr>
<td></td>
<td>Chloroquine phosphate 150 mg/d for 3-9 months</td>
<td>3</td>
<td>1 case of temporary response (33.3%) 2 cases with no effect on progression of alopecia (66.6%)</td>
</tr>
<tr>
<td></td>
<td>Moderate-potency topical corticosteroid</td>
<td>9</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>2% minoxidil solution</td>
<td>2</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>Intralesional corticosteroid</td>
<td>1</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td>Naz et al 2003</td>
<td>0.05% fluocinolone acetonide cream twice daily for 1 year</td>
<td>1</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>Oral prednisone 1 mg/kg/d for 3 months</td>
<td>1</td>
<td>Hair-loss was halted, though it did continue after suspension of the oral prednisone</td>
</tr>
<tr>
<td></td>
<td>2% minoxidil solution twice daily for 6 months</td>
<td>1</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>0.025% triamcinolone acetonide cream twice daily for 6 months</td>
<td>1</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td>Vaisse et al 2003</td>
<td>Topical corticosteroid (potency I-II) for 2-16 months</td>
<td>8</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>Topical corticosteroid (potency I-II) + hydroxychloroquine</td>
<td>2</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>Topical corticosteroid (potency I-II) + chloroquine</td>
<td>2</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>Topical corticosteroid (potency I-II) + 2% minoxidil solution</td>
<td>1</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>2% minoxidil solution</td>
<td>2</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>Acitretin 30-40 mg/d for 3-6 months</td>
<td>4</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td></td>
<td>Oral prednisone 0.5 mg/kg/d for 6-18 weeks</td>
<td>2</td>
<td>No therapeutic effect</td>
</tr>
<tr>
<td>Moreno-Ramirez et al 2005</td>
<td>Intralesional triamcinolone acetonide 20 mg/mL (1 mg/2 cm², 1/10 dilution on eyebrows) every 3 months</td>
<td>9</td>
<td>Rapid stabilization of frontal recession in 4 patients (44%)</td>
</tr>
<tr>
<td></td>
<td>Intralesional triamcinolone acetonide + finasteride 2.5 mg/d + 5% minoxidil solution twice daily</td>
<td>7</td>
<td>Rapid stabilization of frontal recession in 1 patient (14%)</td>
</tr>
<tr>
<td></td>
<td>Regimen applied to patients with associated androgenetic alopecia</td>
<td></td>
<td>General improvement in 6 patients (86%) with increased hair density, but with no effect on the degree of frontal fibrosing alopecia</td>
</tr>
<tr>
<td>Tosti et al 2005</td>
<td>Intramuscular triamcinolone acetonide 40 mg every 3 weeks + 2% minoxidil solution twice daily</td>
<td>3</td>
<td>No response. Slow progression of alopecia</td>
</tr>
<tr>
<td></td>
<td>Finasteride 2.5 mg/d + 2% minoxidil solution twice daily</td>
<td>8</td>
<td>Progress halted in 4 patients (50%) after between 12 and 18 months of treatment No response. Slow progression of alopecia in 4 patients (50%) after between 6 and 9 months of treatment</td>
</tr>
</tbody>
</table>
reviewed, 60% were treated with corticosteroid monotherapy
and 73% of patients were treated with corticosteroids alone
or in combination with another drug.

**Systemic Corticosteroids**

Administration of oral prednisone halted hairline recession
in 42.9% of patients treated. However, in the cases where
this treatment was beneficial, the disease continued to progress
when administration of corticosteroids was suspended. The
administered dosages were 0.5–1 mg/kg/d for a period of 3
to 18 months.3,4 However, administration of 25–50 mg/d
of oral prednisone in short cycles of 1 month was the regimen
that provided the greatest, if transient, benefit.1,2

**Topical Corticosteroids**

As shown in Table 5, topical administration of medium to
high-potency corticosteroids (I–II) did not halt progression
of the alopecia in any of the patients in the reviewed studies.
The treatment regimens described consisted of administering
0.05% fluocinolone acetonide or 0.025% triamcinolone
acetonide twice daily for 2 to 6 months. The studies reviewed
reported no adverse effects due to the topical application
of corticosteroids despite prolonged administration on
moderately atrophied skin, which is potentially more
sensitive to the local effects of corticosteroids.1-6

**Intralesional Corticosteroids**

Intralesional administration of triamcinolone acetonide
provided a response rate of 40% (Table 5). This response
was obtained in patients in whom the biopsy revealed an
active inflammatory infiltrate. The administration regimen
consisted of triamcinolone acetonide at a dose of 1 mg/cm²
every 3 months using a solution of 20 mg/mL for
administration in the scalp and a solution of 2 mg/mL
(10%) for administration in the eyebrows, as this location
is more susceptible to atrophy.5

Once alopecia has advanced to the fibrotic phase,
intralesional corticosteroids provide no benefit and may
even worsen the fibrosis and atrophy that characterize the
advanced stages of frontal fibrosing alopecia.

**Minoxidil**

The pathogenic mechanism of frontal fibrosing alopecia
(inflammation and fibrosis) is different from that of
androgenetic alopecia (miniaturization). Nevertheless,
minoxidil, which has a known effect on reducing the rate
of follicle miniaturization, has been tried in patients with
frontal fibrosing alopecia. Approximately a third of the
patients in the reviewed studies received minoxidil.

A 2% solution of minoxidil was applied twice daily for
6 months as monotherapy in 5 patients and did not slow
the progression of the frontal alopecia.1-6

**Finasteride**

Finasteride is an inhibitor of 5α-reductase and prevents the
follicular miniaturization that characterizes androgenetic
alopecia by blocking the conversion of testosterone to
dihydrotestosterone. For this reason, in the cases where
finasteride was administered to patients with frontal fibrosing
alopecia, the improvement obtained was related to the
improvement in the level of the associated androgenetic
alopecia.5,6 Finasteride was not used as monotherapy in any
of the patients with frontal fibrosing alopecia. Of the patients
who did receive this treatment (n = 15), 8 were administered
2.5 mg/d of finasteride in association with a 2% solution of
minoxidil. Progression of the alopecia was halted in 4 of these
patients (50%) after 12-18 months, whereas the other 4
patients (50%) showed no response. Administration of the
same dosage of finasteride in association with minoxidil and
intralesional triamcinolone acetonide to 7 patients provided
general improvements with increased hair density in 86% of
patients (n = 6) but had no effect on the degree of frontal
fibrosing alopecia.5,6 There is no risk of feminization of male
fetuses in these cases due to the mean age of the patients
with frontal fibrosing alopecia (more than 60 years of age).

**Antimalarial Drugs**

In the original studies by Kossard et al,1,2 administration
of 150 mg/d of chloroquine phosphate for 3-9 months in
3 patients obtained a temporary response in 1 patient. In
the study by Vaisse et al,4 the association of
hydroxychloroquine and topical corticosteroids provided
no response in either of the 2 patients treated.

**Other Treatments**

There are anecdotal cases in which a small number of patients
with frontal fibrosing alopecia were treated with griseofulvin,
isotretinoin, tacrolimus, pimecrolimus, cyclosporine etc.
These treatments were not shown to be effective in halting
the progression of alopecia in any of the cases.

**Conclusions**

1. Diagnosis of frontal fibrosing alopecia is primarily clinical
in the case of a middle-aged or elderly woman with a
receding frontal hairline.
2. The existence of other manifestations (scarring alopecia, eyebrow alopecia, axillary alopecia, or perifollicular papules) will allow for greater certainty in the clinical diagnosis and differential diagnosis with other forms of scarring alopecia.

3. Histopathology is characterized by a perifollicular lymphocytic infiltrate surrounding the upper parts of the follicle, with lamellar fibrosis in advanced stages.

4. There are no therapeutic options to date that have proven to be effective with an appropriate level of evidence in the treatment of frontal fibrosing alopecia.

Conflicts of Interest
The authors declare no conflicts of interest.

References


