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Types and Treatment of Hair Loss in Men and Women

Emma Coleman, RGN

In this article, the author focuses on 4 common hair loss disorders that occur in both men and women. The author discusses research related to androgenetic alopecia, telogen effluvium, alopecia areata, and scarring alopecia and provides details on how to approach and manage these diseases according to patient gender. There are a range of tools and tests that can assist with the diagnostic process and help ensure that relevant and high standards of patient care are maintained. In some cases, no medical intervention is always a treatment option. However, appropriate medical treatments, although still relatively limited in some cases, are safe and have proven efficacy. Hair loss has immense emotional and psychological impact in both genders, and it is always important to consider this when planning hair loss management pathways.

Hair loss can be classified by its cause and presentation in all cases and, in some cases, by patient gender. In this article, the author focuses on four common hair loss disorders that occur in both men and women. The author discusses research findings related to androgenetic alopecia (AGA), telogen effluvium (TE), alopecia areata (AA), and scarring alopecia (SA) and provides details on how to approach and manage these diseases according to patient gender. Notably, there is some overlap in the causes and symptoms of the disorders, which highlights the need for obtaining a clear differential diagnosis (Malkud, 2015). There are a range of tools and tests that can assist with the diagnostic process and help ensure that relevant and high standards of patient care are provided and maintained. In some cases, the patient may choose not to proceed with medical intervention. However, appropriate medical treatments, although still relatively limited in some cases, are safe and have proven efficacy. Hair loss has immense emotional and psychological impact in both genders. It is always important to consider this when planning hair loss management pathways (Ruiz-Doblado, Carrizosa, & Garcia-Hernandez, 2003). Following is a discussion of common hair loss disorders that occur in both men and women.

ANDROGENETIC ALOPECIA

Androgenetic alopecia, as its name suggests, is thought to be androgen-dependent (National Institute for Health and Care Excellence [NICE], 2016a, 2016b). This condition presents as diffuse hair loss in both genders but is more likely to occur in postmenopausal women, affecting about 33% of genetically predisposed Caucasian women 70 years or older. In women with AGA, hair loss usually affects the top of the scalp (NICE, 2016b). As many as 58% of men aged 30–50 years may experience AGA. In men, hair loss typically starts with bitemporal hairline recession and hair thinning at the crown (i.e., vertex) and frontal parietal areas (NICE, 2016a). According to the Hamilton–Norwood Scale (Norwood, 1975), the amount of hair loss increases with age. Men with Grade I–III hair loss can benefit from medical intervention. Men with Grade IV–VI hair loss generally show good response to hair transplant procedures (Shankar, Chakravarti, & Shilpakar, 2009). In both men and women, the underlying pathological process involves pigmented terminal hairs gradually being replaced by smaller, less pigmented hairs with a similar appearance to the short, thin, barely noticeable vellus hairs that develop on the body during childhood (NICE, 2016a, 2016b). See Table 1 for signs and symptoms, diagnostic tools, investigations, and management of AGA in men and women.

TELOGEN EFFLUVIUM

Telogen effluvium is a nonscarring form of hair loss. It generally occurs about 3 months after a triggering event when up to 70% of the anagen-phase hairs are precipitated into telogen hairs, with a bulb or club at their tip. After a few months, new hair pushes the club hairs up and out. This disorder is usually self-limiting, lasting for about 6 months before the hair regrows, provided the trigger is not repeated. There is a diffuse, but temporary loss of these hairs in both genders, and there is often a noticeable change in fingernail growth that occurs at the same time (DermNet NZ, 1997; Trüeb, 2008).

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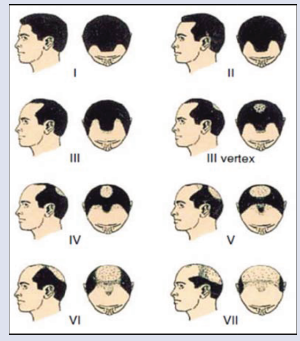
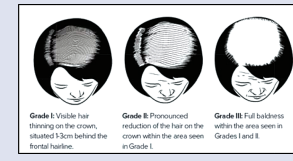
TABLE 1 Androgenetic Alopecia

	Men	Women
Signs and symptoms	Typically presents with bitemporal hairline recession, with hair thinning at the crown and frontal parietal areas. In approximately 10% of cases, male AGA presents with a female pattern of hair loss. The normal hair may be replaced by thin, vellus hairs (NICE, 2016a).	Usually appears as diffuse hair loss affecting the crown of the scalp while the frontal hairline is retained. Complete baldness is rare, and normal hair is replaced by thin, vellus hairs (NICE, 2016b).
Diagnostic tools	Diagnosis is usually made clinically through observation and history (NICE, 2016a). The Hamilton–Norwood Scale may be used to assess the degree of hair loss:  The Hamilton–Norwood Scale of male pattern baldness. From "Male Pattern Baldness; Classification and Incidence," by O. T. Norwood, 1975. *Southern Medical Journal, 68*(11), pp. 1359–1365. Copyright 1975 by the Southern Medical Association. All rights reserved. Reprinted with permission.	Diagnosis is usually made clinically through observation and history (NICE, 2016b). The Ludwig Scale may be used to assess degree of hair loss:  The Ludwig Scale. From "Case Study: Treating Female-Patterned Hair Loss," by L. Godfrey, 2016, *Aesthetics, 3*(8), pp. 50–53. Copyright 2016 by Aesthetics. All rights reserved. Reprinted with permission.

Investigations	• *History*:
	  ○ Assess the timing/pattern of hair loss, including speed, onset, areas affected, and progression.
	  ○ Systemic disease of the endocrine system (rare) and/or infection could lead to AGA in males.
	  ○ Medications such as antidepressants, chemotherapy drugs, anabolic steroids, and anticoagulants may also cause AGA.
	  ○ Ascertain whether there is a familial history of alopecia.
	  ○ Assess for extreme dietary habits or rapid weight loss.
	  ○ Evaluate the usual hair care routine of the patient.
	• Perform the pull test to help differentiate between AGA and TE by grasping approximately 50–100 hairs and pulling along the shaft. The test is positive if more than three hairs come away.
	• Generally, male AGA is not caused by endocrine imbalance; however, consider ordering laboratory tests (e.g., iron levels, thyroid function, ferritin, complete blood cell count) if the underlying disease is present or TE is suspected (Blume-Peytavi et al., 2011; NICE, 2016a).
		• *History*:
		  ○ Evaluate the patient for the possible underlying disease (e.g., iron deficiency, infection, hypothyroidism).
		  ○ Medications including antidepressants, anabolic steroids, antithyroid agents (e.g., carbimazole), and chemotherapy drugs can lead to AGA.
		  ○ Assess for extreme dietary habits or rapid weight loss.
		• Examine scalp for any inflammation, scarring, papules, pustules, etc., that could point to folliculitis.
		• Although not necessary, performing laboratory tests for thyroid function, complete blood cell count, and ferritin level may enhance the clinical picture and help differentiate AGA from other types of hair loss.
		• Consider basic endocrine investigations if there are features of elevated androgens present (e.g., hirsutism, acne vulgaris).
		• Trichoscopy (20- and 70-fold magnification) may be used to differentiate between AGA and TE in women (NICE, 2016b; Oakley, 2014; Rakowska, Slowinska, Kowalska-Oledzka, Olszewska, & Rudnicka, 2009).

(*continues*)

TABLE 1 Androgenetic Alopecia (*Continued*)

	Men	Women
Management	• No treatment is an option in mild cases. • *Medications*: ◦ Topical minoxidil 2% or 5% twice daily. Educate the patient that irritation may occur when used at higher concentrations. Assess treatment efficacy after 6 months. ◦ Oral finasteride 1 mg daily. Assess treatment efficacy at 6 and 12 months. Risk of side effects includes reduced libido, erectile dysfunction, and ejaculation problems, which occur in 1%–2% of men (NICE, 2016a). • Evidence suggests that these medications are more effective in early-onset AGA. If no benefit obtained by 12 months, stop treatment and refer the patient to a dermatologist. Both treatments must be taken indefinitely to maintain benefit. • *Education*: Provide information about protection from sun and cold. Providing information about prosthetics, such as wigs and hair pieces, may also be helpful. • *Psychological effects*: Hair loss can negatively affect confidence levels in men, and the level of effect is not necessarily linked to the amount of hair loss (Han et al., 2012; NICE, 2016a).	• No treatment is always an option in mild cases. • *Medications*: ◦ Topical minoxidil 2% or 5% twice daily. Educate the patient that irritation may occur if used at higher concentrations. Assess treatment efficacy after 6 months. ◦ Oral finasteride 1 mg daily. It should not be prescribed for women of childbearing age (NICE, 2016b). • Evidence suggests that these medications are more effective in early-onset AGA. If no benefit obtained by 12 months, stop treatment and refer the patient to a dermatologist. Both treatments must be taken indefinitely to maintain benefit. • *Education*: Provide information about protection from sun and cold. Providing information about prosthetics, such as wigs and hair pieces, may also be helpful. • *Psychological effects*: Hair loss can negatively affect self-esteem, leading to feelings of isolation, embarrassment, and ultimately depression and anxiety (NICE, 2016b; Van Zuuren, Fedorowicz, & Schoones, 2016).

Note. AGA = androgenetic alopecia; TE =telogen effluvium.

Known triggers for TE include stress, shock, acute fever, invasive surgery, severe infection or trauma, thyroid disorder, low protein diet or extreme dieting, iron deficiency, and certain medications (e.g., chemotherapy drugs, β-blockers, retinoids, anticoagulants, propylthiouracil, carbamazepine, immunizations; Hughes & Saleh, 2019). Hormonal changes in pregnancy, particularly a decrease in estrogen levels, can lead to postpartum TE (MedicineNet.com, n.d.); however, the studies supporting this information are small and additional investigation is necessary to establish a connection (Mirallas & Grimalt, 2016). A subtype of TE, known as chronic TE, occurs more commonly in middle-aged women with long, thick hair (Cunliffe, 2019). See Table 2 for signs and symptoms, diagnostic tools, investigations, and management of TE in men and women.

ALOPECIA AREATA

Alopecia areata presents as nonscarring hair loss. In AA, the hair loss occurs as a solitary patch or as several well-demarcated patches (Camacho, 1997; Tan, Tay, Goh, & Chin Giam, 2002). This type of hair loss occurs when the hairs are prematurely converted from the growth (i.e., anagen) phase to the loss (i.e., telogen) phase (NICE, 2018). The specific cause of AA is unknown, although 20% of people with AA have a positive family history (Messenger, McKillop, Farrant, McDonagh, & Sladden, 2012). Alopecia areata is associated with other autoimmune conditions (e.g., thyroid disease). Men with relevant family history and boys 10 years or younger are more likely to experience AA, but the incidence is generally higher in women, with peak occurrence between 10 and 20 years of age (Lundin et al., 2014). Hair regrowth is common in clients with only minor hair loss and usually regrows within a year; however, regrowth can be unpredictable (NICE, 2018). Some researchers have reported that emotional stress triggers AA (Baker, 1987), whereas other researchers completely refute this (Colón, Popkin, Callies, Dessert, & Hordinsky, 1991). Additional research related to AA is warranted. See Table 3 for signs and symptoms, diagnostic tools, investigations, and management of AA in men and women. Figure 1 provides a treatment protocol for AA in different age groups.

SCARRING ALOPECIA

Scarring alopecia is irreversible and is more commonly seen in women, especially European Caucasian women with a mean age of 51–53 years (Villablanca, Fischer, García-García, Mascaró-Galy, & Ferrando, 2017). Scarring alopecia is divided into two subgroups.

Primary scarring alopecia (PSA) occurs due to an underlying disease (Patterson, 2014) that leads to a lack of follicular ostia and replacement of hair follicles with fibrous tissue. It is associated with conditions that include chronic cutaneous lupus erythematous, pseudopelade of Brocq, lichen planopilaris, folliculitis decalvans, and dissecting folliculitis. In some cases, the type of underlying disease can be identified by the type of inflammatory

TABLE 2 Telogen Effluvium

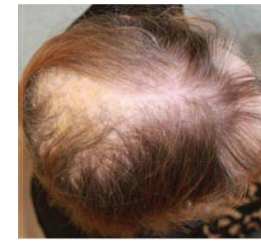
	Men	Women
Signs and symptoms	• Yun and Kim (2007) examined TE presentation in patients undergoing chemotherapy. Among 20 men with patterned hair loss, occipital hairlines were preserved in 50% ($n = 10$), frontal hairlines in 15% ($n = 3$), and both occipital and frontal hairlines in 35% ($n = 7$). • Generally, TE presents as a diffuse loss of club hair in the telogen growth phase around 12 weeks following a trigger (DermNet NZ, 1997). • Trüeb (2008) reported that 9% of males experienced trichodynia associated with TE. • The time of the shock or illness may be denoted by stunted nail growth and a ridge or Beau line may appear around the time of hair shedding (DermNet NZ, 1997). • Hair regrowth is generally spontaneous after 6–9 months (DermNet NZ, 1997).	• Yun and Kim (2007) examined TE presentation in patients undergoing chemotherapy. Among 25 women with patterned hair loss, occipital hairlines were preserved in 8% ($n = 2$), frontal hairlines in 40% ($n = 10$), and both occipital and frontal hairlines in 52% ($n = 13$). • Generally, TE presents as a diffuse loss of club hair in the telogen growth phase around 12 weeks following a trigger (DermNet NZ, 1997). • TE appears to be more common in women, although this may be because women are more likely to report and seek treatment of hair loss than men (Malkud, 2015). • Chronic TE has a higher incidence in middle-aged women with long hair (Cunliffe, 2019), with shedding seen in areas normally spared by AGA, especially the supra-auricular area (Rebora, 2016). • Willimann and Trüeb (2002) found that women with TE reported a higher incidence of trichodynia and discomfort originating from the hair and scalp than men. • Up to 90% of women experience postpartum TE due to hormonal changes (MedicineNet, n.d.). Although lactation seems to influence the hair's anagen rate, the studies conducted thus far are too small to provide any solid evidence about a connection with TE (Mirallas & Grimalt, 2016). • The time of the shock or illness may be denoted by stunted nail growth and a ridge or Beau line may appear around the time of hair shedding (DermNet NZ, 1997). • Hair regrowth is generally spontaneous after 6–9 months (DermNet NZ, 1997).  Telogen effluvium secondary to thyrotoxicosis. From *Alopecia—An Overview*, by T. Cunliffe, 2019. Retrieved from http://www.pcds.org.uk/clinical-guidance/alopecia-an-overview. Image courtesy of Primary Care Dermatology Society.
Diagnostic tools	The following classification may be helpful in highlighting the cause and severity of TE in both men and women: 1. *Premature teloptosis*: Often due to proteolysis, triggered by endogenous or exogenous factors (e.g., use of shampoos containing minoxidil, presence of seborrheic dermatitis). 2. *Collective teloptosis*: Molt-like shedding may occur due to physiological or drug-induced conditions in which the hair cycles are synchronized. Most often seen in newborns and postpartum women, also associated with certain drugs, including contraceptives, minoxidil, and finasteride. 3. *Premature entry into the telogen phase*: Caused by interruption of keratinocyte cell division in the hair matrix. The anagen phase of hair growth is disrupted, accelerating hairs to the telogen stage. Likely causes are drugs (e.g., chemotherapy), dietary insufficiency, and autoimmune disorders. Antithyroperoxidase antibodies and Hashimoto's thyroiditis are present in up to 60% of cases (Rebora, 2016).	

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TABLE 2 Telogen Effluvium (*Continued*)

	Men	Women
Investigations	• *Wash test*: A reliable pathway for diagnosis of TE. The patient is instructed to wash his or her hair 5 days after the last shampoo in a sink with its drain covered by gauze. The hair entrapped in the gauze is then counted (Amin & Sachdeva, 2013). • *Pluck test (Trichogram)*: If there are 25% or more telogen hairs present, the test is positive. Telogen hairs are hairs that have tiny bulbs without sheaths at their roots (Amin & Sachdeva, 2013). • *Punch biopsy with histopathological analysis*: Although not necessary, a biopsy shows the number of telogen follicles and can aid in diagnosis (Malkud, 2015). Findings in TE are best seen in transverse sections of a punch biopsy. If 25% of the follicles are in the telogen phase, this confirms the diagnosis of TE (Amin & Sachdeva, 2013). • *Laboratory testing*: Chronic TE sometimes has a metabolic cause, such as hypothyroidism. If symptoms of thyroid malfunction are present, a thyroid function test should be performed. Iron levels should be assessed with complete blood cell count, hemoglobin, and ferritin testing. If syphilis is suspected, a rapid plasma or venereal disease research laboratory test should be performed (Hughes & Saleh, 2019).	
Management	• *Psychological management*: ○ Patients should be reassured that normal grooming of hair will not increase hair loss or prevent regrowth (Hughes & Selah, 2019). • *Lifestyle*: Cigarettes contain heavy metals that can worsen TE; therefore, smoking cessation may be helpful (British Association of Dermatologists [BAD], 2016). • Educate patients about normal hair cycles and the relationships between triggers and timing of hair loss (Malkud, 2015).	• *Psychological management*: ○ Women are more likely to have lowered quality of life and restricted social contacts as a result of TE (Dinh & Sinclair, 2007). ○ Patients should be reassured that normal grooming of hair will not increase hair loss or prevent regrowth (Hughes & Selah, 2019). • *Lifestyle*: Cigarettes contain heavy metals that can worsen TE; therefore, smoking cessation may be helpful (BAD, 2016). • Educate patients about normal hair cycles and relationships between triggers and timing of hair loss (Malkud, 2015).
	• Hair transplant has no role in the management of TE as it is a temporary disorder (Malkud, 2015). • Once the causative issue has been identified and withdrawn, the TE should also reside. • Although topical minoxidil has not been proven to promote hair regrowth in cases of TE, it may help patients who wish to take an active role in their treatment (Hughes & Selah, 2019).	

Note. AGA = androgenetic alopecia; TE = telogen effluvium.

infiltrate around the hair follicles (Olsen et al., 2003; Villablanca et al., 2017). Figure 2 provides a classification system for identifying the underlying disease associated with PSA, which can help determine treatment and management for both men and women.

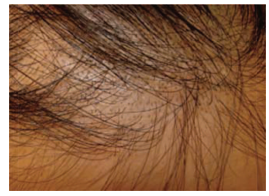
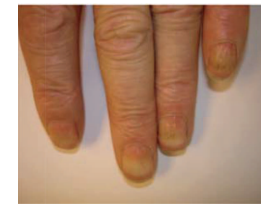
Secondary scarring alopecia (SSA) is so named because it results from exogenous factors such as trauma caused by burns or radiation or by endogenous inflammatory processes caused by disorders such as sarcoidosis, pemphigus vulgaris, or scleroderma (Villablanca et al., 2017). Although controversial, there is evidence to suggest that central centrifugal cicatricial alopecia (CCCA) is a type of SSA largely caused by hair treatments such as straightening with hot irons and using products with chemicals that damage hair follicles. This type of alopecia is seen predominantly in women of Afro-Caribbean descent (Nicholson, Harland, Bull, Mortimer, & Cook, 1993; Sperling & Sau, 1992). There is some evidence to suggest that CCCA possesses an autosomal mode of inheritance (Diova, Jordaan, Sarig, & Sprecher, 2014). One small study showed some correlation between the incidence of CCCA and diabetes mellitus (Kyei, Bergfeld, Piliang, & Summers, 2011). To correctly classify CCCA, more large-scale and widespread investigation is necessary.

Figure 3 provides a two-step algorithm that can be used with both scarring and nonscarring forms of alopecia where the presence or absence of follicular miniaturization and raised or normal catagen/telogen counts are used as markers for differential diagnosis. See Table 4 for signs and symptoms, diagnostic tools, investigations, and management of SA in men and women.

DIAGNOSTIC TOOLS

Differentiation in the diagnostic process is difficult but essential and is achieved with a variety of tools including biopsy with histopathology. Histopathology of AGA will show prominent sebaceous glands, miniaturization of existing hair follicles, and reduced follicular diameter (Soeprono, 2012a). Telogen effluvium histopathology will show abnormally high telogen follicles with empty follicular sheaths known as "stele" (Malkud, 2015; Soeprono, 2012b). Alopecia areata histopathology typically presents with a "swarm of bees" appearance caused by inflammatory infiltrate around terminal hair follicles (Amin & Sachdeva, 2013). Scarring alopecia histopathology will be identical in both genders and is characterized by collagen cells becoming translucent and highlighted by wide, tree trunk-like tracts

TABLE 3 Alopecia Areata

	Men	Women
Signs and symptoms	• Tends to be more severe in men (Cole, 2018). • Men are more likely to have a familial history and may be diagnosed with AA when younger than 10 years (Lundin et al., 2014). • More large-scale studies investigating gender association are needed.	• Incidence is generally higher in females (2.3:1 female vs. male), with peak diagnosis at 10–20 years of age. • Women are more likely to have morbid nail symptoms and autoimmune diseases, usually thyroid disorders (Lundin et al., 2014). • More large-scale studies investigating gender association are needed.
	 Exclamation mark hairs. From "Alopecia Areata: Clinical Presentation, Diagnosis, and Unusual Cases," by A. M. Finner, 2011, *Dermatologic Therapy, 24*(3), pp. 348–354. Copyright 2011 by Wiley Periodicals, Inc. All rights reserved. Reprinted with permission.	 AA of the nails. From "Alopecia Areata: Clinical Presentation, Diagnosis, and Unusual Cases," by A. M. Finner, 2011, *Dermatologic Therapy, 24*(3), pp. 348–354. Copyright 2011 by Wiley Periodicals, Inc. All rights reserved. Reprinted with permission.
	• Clinical features are the same in both genders. AA most commonly presents as a sudden loss of hair in localized areas. The patch is usually round or oval-shaped and well demarcated at the borders. It may be a solitary patch (AA monolocularis) or numerous patches (AA multilocularis) (Camacho, 1997; Tan, Tay, Goh, & Chin Giam, 2002). • The patch of alopecia usually has a distinct border, with normal hair demarcating the periphery of the lesion. • The scalp is the most common site affected by AA (90%). • Scalp and body hair such as eyebrows, eyelashes, beard, underarm hair, and pubic hair may be affected (alopecia totalis), as well as the entire body (alopecia universalis) (Camacho, 1997; Tan et al., 2002). • Clinicians should look for "exclamation point hairs" surrounding the patches. These are hairs that are thick at the top and become narrower along the length of the strand closer to the base with a root at the bottom (Cline, 1988). • Regrowing hair often lacks pigment, so it is white or blonde in color (Finner, 2011). • Nail changes are seen in 10%–66% of cases. ◦ Red-spotted lunula and periungual erythema are a sign of acute nail involvement. ◦ Small shallow pits and trachyonychia are typical (Olsen, 2003).	
Diagnostic tools	Diagnosis is usually by clinical observation in both genders, although there are diagnostic tools that can be used to help ascertain disease severity: • One scale presents the clinical signs of AA as follows: ◦ *Mild*: Three or fewer patches of alopecia with a widest diameter of less than 3 cm. ◦ *Moderate*: More than three patches or a patch greater than 3 cm at the widest diameter without alopecia totalis or universalis. ◦ *Severe*: Alopecia totalis or alopecia universalis. • *Ophiasis*: Severe alopecia where the loss of hair occurs in the shape of a wave at the circumference of the head (Kavak, Baykal, Özarmağan, & Akar, 2001). • SALT ◦ The scalp is divided into four areas. ◦ The percentage of hair loss in any of the four areas is calculated using the following formula: % of hair loss × % of surface area of the scalp in that area. ◦ The areas are marked out as follows: ■ Vertex—40% (0.4) of scalp surface area ■ Right profile of scalp—18% (0.18) of scalp surface area ■ Left profile of scalp—18% (0.18) of scalp surface area ■ Posterior aspect of scalp—24% (0.24) of scalp surface area. ◦ The SALT score is the sum of percentages of hair loss in all the aforementioned areas (Price & Gummer, 1989).	

(*continues*)

TABLE 3 Alopecia Areata (*Continued*)

	Men	Women
Investigations	• Explore familial history. • Most cases in both men and women can be diagnosed clinically, requiring no investigation, although there are some protocols that may be helpful when diagnosis is in doubt (Messenger et al., 2012). • *Pull test*: Evaluates diffuse scalp hair loss. About 40–60 hairs are gently pulled in three different scalp areas. A positive result occurs when 10 or more hairs are obtained. • *Pluck test*: The individual pulls out hairs by the roots, which are then examined under a microscope to determine the phase of growth and differentiate between telogen, anagen, or systemic disease. Anagen hairs have sheaths attached to their roots. Anagen effluvium shows raised broken hairs and a decrease in hairs at telogen growth phase. • *Scalp biopsy and histopathological analysis*: These are carried out when alopecia is present, but diagnosis is unclear. The biopsy allows for differentiation between scarring and nonscarring forms. Hair samples are taken from areas of inflammation, usually around the border of the patch. Biopsy results will depend on the severity of AA, rather than the patient's age, race, or gender (Igarashi, Morohashi, Takeuchi, & Sato, 1981). ◦ In the subacute stage, increased catagen and telogen hairs are seen. ◦ In the acute stage, terminal hairs are encompassed by bulbar lymphocytes or the "swarm of bees" appearance. ◦ In the chronic stage, decreased terminal and increased miniaturized hairs are found, with variable amounts of inflammation. • *Daily hair counts*: These are carried out in cases of negative pull test and done by counting the number of hairs lost each day. Hairs should be counted after the first morning combing or washing. Hairs are collected in a clear plastic bag for 14 days. The number of strands is recorded. If the hair count is more than 100 per day, this is abnormal (except after shampooing, where normal hair counts may reach up to 250). • *Trichoscopy*: A noninvasive hair and scalp analysis may be performed using a handheld dermoscope or a video dermoscope. In AA, trichoscopy will highlight yellow dots (i.e., hyperkeratotic plugs), tiny exclamation mark hairs, and black dots (i.e., destroyed hairs in follicles) (Amin & Sachdeva, 2013).	
Management	• Management tends to be adjusted according to age rather than sex. • There is no definitive cure, and the main gender difference in management will be in the use of appropriate prosthetic types (e.g., women may prefer a head scarf to a hair piece) (NICE, 2018). • *Intralesional corticosteroids*: These are indicated as first-line treatment in AA, with hair loss effecting less than 50% of the scalp. 10 mg/mL of triamcinolone acetonide is injected with a 0.5-in. needle in 0.1-ml injections, approximately 1 cm apart. Review treatment effect at 4–8 weeks; repeat every 4–6 weeks if needed (Pascher, Kurtin, & Andrade, 1970). • *Topical corticosteroids*: Painless and present a good option with children (e.g., fluocinolone acetonide cream, fluocinolone scalp gel, betamethasone valerate lotion, clobetasol propionate ointment); however, results are sporadic (Amin & Sachdeva, 2013). • *Systemic corticosteroids*: Consider only in severe cases, as the side effects of these drugs may be limiting. One small-scale study involving 20 participants provided evidence that a tapering course of prednisolone improved hair regrowth by more than 25% in 30%–47% (n = 9–14) of the participants with mild to severe AA (Sharma & Gupta, 1999). Another study showed some success using oral mini-pulse therapy with corticosteroids, which is a type of systemic treatment where large amounts of medications are administered to patients in short intervals to achieve stronger medication effects and avoid long-term use. The researchers found the treatment minimized side effects (Pasricha & Kumrah, 1996). • *Glucocorticoids*: These may be prescribed for their anti-inflammatory effects (Ross & Shapiro, 2005). • *Minoxidil*: A topical agent that stimulates follicle proliferation at the root and allows for differentiation above the dermal papilla, independent of its vascular influences (Fiedler, Wendrow, Szpunar, Metzler, & DeVillez, 1990). • *Anthralin*: A topical agent that works by promoting free radical production over the scalp, leading to erythema or pruritus after application of 0.5%–1% once daily over a 2-week period. The treatment is continued for 3–6 months, (Ross & Shapiro, 2005). Anthralin is particularly effective when used in combination with minoxidil (Fiedler et al., 1990). • *Topical immunomodulators*: One recent study assessed the use of inosiplex in nine participants with AA (i.e., totalis). All participants developed enhanced T-cell function, and seven participants displayed significant hair regrowth (Galbraith, Thiers, & Fundenberg, 1984). Additional studies are necessary to further assess efficacy and side effect risks. • *PUVA*: Reduces inflammation around the hair follicles caused by Langerhans and mononuclear cells (Amin & Sachdeva, 2013). One study investigated the effect of PUVA with 26 participants; 54% displayed greater than 90% hair regrowth; those with alopecia areata universalis had more successful outcomes, whereas those with a positive familial history and alopecia areata totalis were less likely to respond to this treatment (Whitmont & Cooper, 2003). • *Oral cyclosporine*: Inhibits T-cell activity; however, its risk for hepatotoxicity, nephrotoxicity, and other side effects may outweigh its benefits (Gupta et al., 1990). • *Topical tacrolimus*: A calcineurin inhibitor that prevents activation of many cytokines in the inflammatory process including interleukin-2, interferon, and tumor necrosis factor. There have been conflicting reports of its efficacy in promoting hair regrowth in AA cases (Amin & Sachdeva, 2013). • *Oral sulfasalazine*: It has immunosuppressive and immunomodulatory effects. One small study with 39 participants showed good hair regrowth in 26% of patients (n = 10) following treatment with 3 g of oral sulfasalazine. A moderate response was seen in 31% of participants (n = 12) (Ellis, Brown, & Voorhees, 2002). Because of its positive safety profile, this drug may present a preferable choice over tacrolimus and long-term steroids.	

Note. AA = alopecia areata; PUVA = psoralen plus ultraviolet light therapy; SALT = Severity of Alopecia Tool.

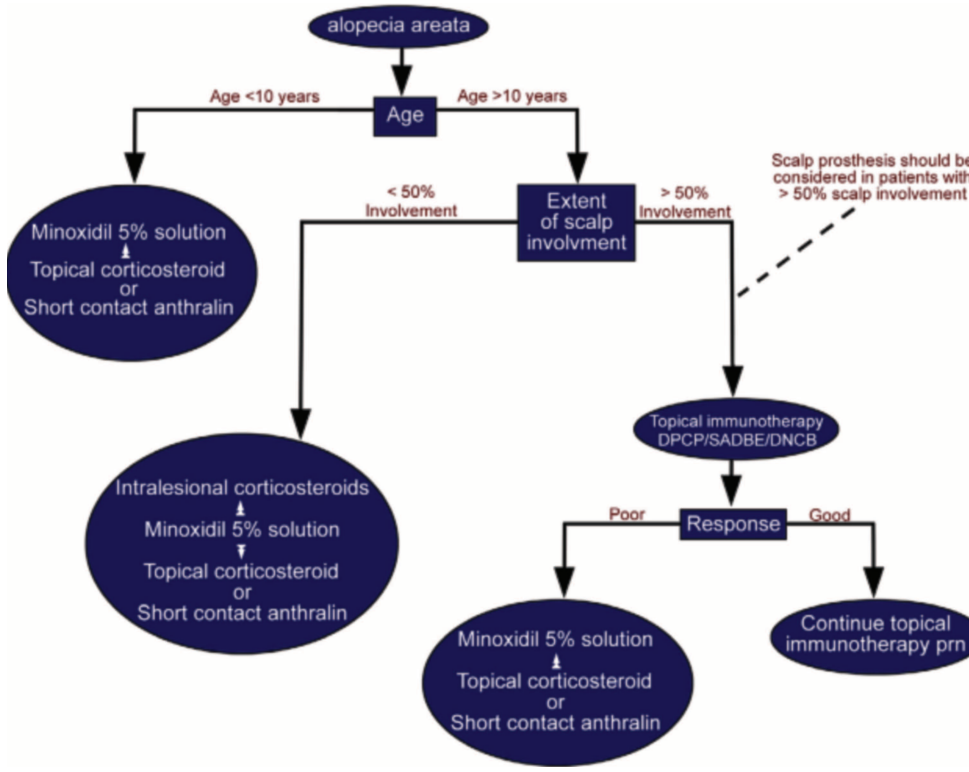


FIGURE 1. Treatment protocol for alopecia areata is licensed under Creative Commons Attribution-NonCommercial-NoDerivs 3.0 Unported [no modifications]. From "Alopecia Areata: A Review," by S. S. Amin and S. Sachdeva, 2013, *Journal of the Saudi Society of Dermatology & Dermatologic Surgery, 17,* pp. 37–45. Copyright 2013 by Wolters Kluwer. Used with permission. This figure is available in color online (www.psnjournalonline.com).

and preservation of the elastin sheaths surrounding these tracts (Blattner, Polley, Ferritto, & Elston, 2013).

The use of dermoscopy or trichoscopy may be invaluable in aiding differential diagnosis (Xu, Liu, & Senna, 2017) as this examines the follicular and interfollicular patterns, as well as the hair shaft characteristics (Jain, Doshi, & Kopkar, 2013). Refer to Table 5 for a comparison of the different histopathology and trichoscopy patterns in these four diseases.

In addition to obtaining a complete personal and family history, various physical tests such as the "pluck" and "wash" tests (Amin & Sachdeva, 2013; Trüeb, 2016) and certain laboratory blood tests can help to specifically identify underlying causes of alopecia (Hughes & Selah, 2019).

PSYCHOLOGICAL EXPLORATION AND MANAGEMENT

Psychological exploration and management must be incorporated in all cases of hair loss. It has been reported that in AA patients, there is a high prevalence of mood, adjustment, depressive, and anxiety disorders, regardless of gender (Ruiz-Doblado et al., 2003). In another study that included women experiencing both scarring and nonscarring forms of alopecia, the researchers found that the women with SA scored higher on the Dermatology Life Quality Index, Hospital Anxiety and Depression Scale, and UCLA Loneliness Scale compared with women with non-SA (Katoulis et al., 2015). In a multinational study that included men experiencing hair loss, the men reported feeling less attractive and confident, and this negatively impacted their social life and increased feelings of depression (Alfonso, Richter-Appelt, Tosti, Viera, & Garcia, 2005). These findings emphasize the need for psychological management of men and women experiencing hair loss.

CONCLUSION

There are some gender differences in the clinical signs and diagnosis pathway and management of hair loss disorders. Scarring and nonscarring forms of alopecia generally have a higher prevalence in adult women; however, this may be due to a tendency for women to visit their specialist and seek help earlier than men. It is possible there may be an increase in the number of men seeking treatment in the future (Malkud, 2015). Women with AA are more likely to have nail symptoms than men. The diagnostic tools for AGA differ by gender, and finasteride as a hair-rejuvenating agent is only safe for male patients with hair loss (NICE, 2016a). Characteristic clinical and

Lymphocyte-associated PSAs	Neutrophil-associated PSAs	Mixed inflammatory PSAs	Nonspecific PSAs

• Chronic cutaneous lupus erythematosus
• Lichen planopilaris
  ○ Classic lichen planopilaris
  ○ Frontal fibrosing alopecia
  ○ Graham Little syndrome
• Classic pseudopelade (Brocq)
• Central centrifugal cicatricial alopecia
• Alopecia mucinosa
• Keratosis follicularis spinulosa decalvans
	• Folliculitis decalvans
	• Dissecting cellulitis/folliculitis
		• Folliculitis (acne) keloidalis
		• Folliculitis (acne) necrotica
		• Erosive pustular dermatosis
			• Idiopathic scarring alopecias with inconclusive clinical and histopathologic findings. May include a variety of end stage inflammatory PSAs (e.g., lichen planopilaris, folliculitis decalvans).

FIGURE 2. Primary scarring alopecias. From "Summary of North American Hair Research Society (NAHRS)-Sponsored Workshop on Cicatricial Alopecia, Duke University Medical Center, February 10 and 11, 2001," by E. A. Olsen, W. F. Bergfeld, G. Cotsarelis, V. H. Prince, J. Shapiro, R. Sinclair, et al., 2003, *Journal of the American Academy of Dermatology, 48*(1), pp. 103–110. Copyright 2003 by Elsevier. Adapted with permission. This figure is available in color online (www.psnjournalonline.com).

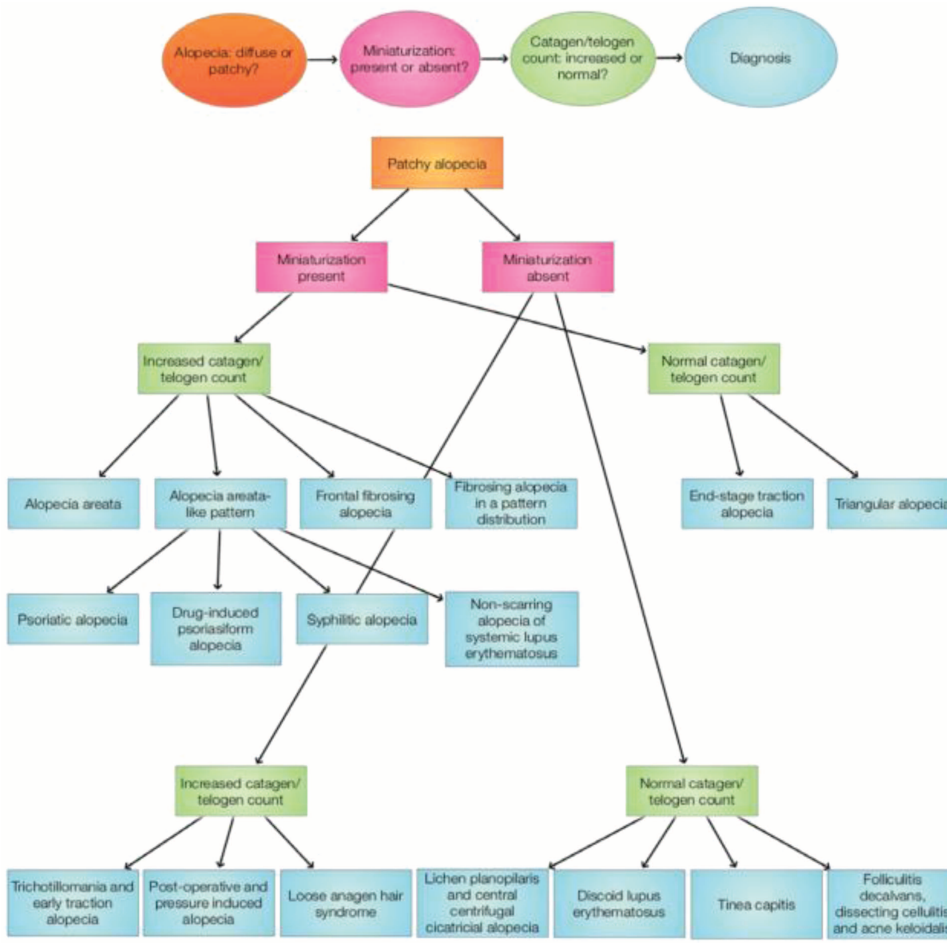
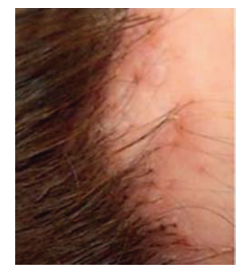


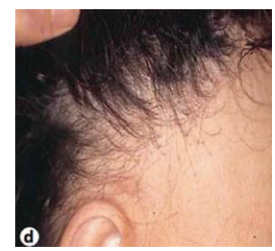
FIGURE 3. Algorithm for differential diagnosis of alopecia. From "Primary Scalp Alopecia: New Histopathological Tools, New Concepts and a Practical Guide to Diagnosis," by A. Kolivras and C. Thompson, 2017, *Journal of Cutaneous Pathology, 44*(1), pp. 53–69. Copyright 2016 by John Wiley & Sons A/S. Reprinted with permission. This figure is available in color online (www.psnjournalonline.com).

TABLE 4 Scarring Alopecia

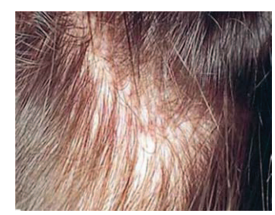
	Men	Women
Signs and symptoms	• Occurs less commonly in men (Villablanca et al., 2017).	• Higher incidence in women, with 93% of cases according to one study (Villablanca et al., 2017). • CCCA is seen almost exclusively in Afro-Caribbean women (Nicholson et al., 1993; Sperling & Sau, 1992).
	• There is no difference in presentation according to gender (Villablanca et al., 2017). • The most common hair loss patterns seen in SA are as follows: 1. Follicular pattern 2. Single large patch 3. Multiple patches 4. Marginal pattern 5. "Footprints in the snow" pattern 6. Folliculitis decalvans pattern 7. Acne keloidalis pattern • Tenderness, itching, and burning are common in CCCA; however, some patients are asymptomatic. • Hair breakage is a frequent early sign, followed by slowly progressing hair loss originating at the vertex (Patterson, 2014). • *Secondary scarring alopecia*: Patients with sarcoidosis will likely present with a lung disorder in addition to cutaneous lesions (Ngan & Stanway, 2002). • Patients with pemphigus vulgaris (often of Indian or Jewish descent) will likely have oral lesions (Ngan & Oakley, 2019). • Scleroderma involves proliferation of collagen and in some cases leads to thickened, damaged skin and hair loss (Ensz, n.d.).	



Follicular pattern PSA associated with lichen planopilaris or folliculitic decalvans.



Marginal pattern PSA associated with frontal fibrosing and traction alopecia.



"Footprints in the snow" pattern often seen in idiopathic PSA.

From "Primary Scarring Alopecia: Clinical–Pathological Review of 72 Cases and Review of the Literature," by S. Villablanca, C. Fischer, S. C. García-García, J. M. Mascaró-Galy, & J. Ferrando, 2017, *Skin Appendage Disorders, 3*, pp. 132–143. Copyright 2017 by S. Karger AG, Basel. All rights reserved. Reprinted with permission.

(*continues*)

TABLE 4 Scarring Alopecia (*Continued*)

	Men	Women
Diagnostic tools	• Assess full clinical history and perform physical examination for signs of the underlying disease, which aids in differentiating between PSA and SSA (Villablanca et al., 2017; Xu et al., 2017). • Assess use of hair products as a possible contributor in men (Sperling & Sau, 1992).	• Assess full clinical history and perform physical examination for signs of the underlying disease, which aids in differentiating between PSA and SSA (Villablanca et al., 2017; Xu et al., 2017). • Assess use of hair products and tools (e.g., chemical products or treatments, straightening irons) and to rule out CCCA (Nicholson et al., 1993; Sperling & Sau, 1992).

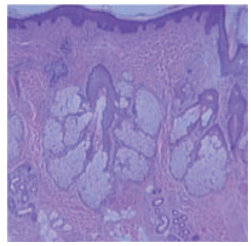
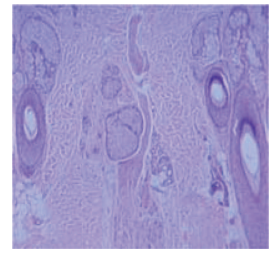
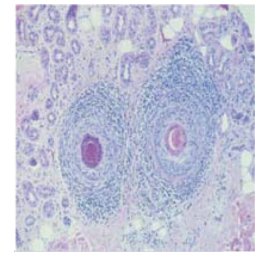
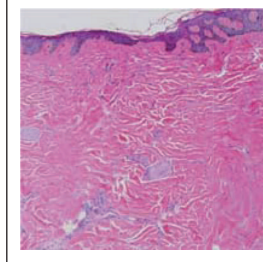
	• PSA poses a diagnostic challenge.
	  ◦ Analysis of the following types of inflammatory infiltrate can be used for differential diagnosis:
	  ▪ Lymphocytic
	  ▪ Neutrophilic
	  ▪ Mixed Inflammatory
	  ▪ Idiopathic
	  ◦ This method can be used across both genders and will aid identification of the underlying disease and therefore management and treatment (Villablanca et al., 2017).
	  ◦ Another helpful diagnostic tool for both scarring and nonscarring forms of alopecia is a two-step algorithm where the presence or absence of follicular miniaturization and raised or normal catagen/telogen count are markers for differential diagnosis (Kolivras & Thompson, 2016).
	• SSA may be easier to identify clinically due to the presence of direct triggers such as recent trauma (Villablanca et al., 2017) and other disease-associated symptoms.	

Investigations	• Clinical observation alone is not sufficient for diagnosis (Villablanca et al., 2017). • *Biopsy*: Taken from the edge of an active patch of alopecia at an early stage of the disease aids accurate diagnosis (Villablanca et al., 2017). In some cases, a biopsy may be used to highlight inflammatory cell infiltrate surrounding the infundibulum (Patterson, 2014), such as perifollicular lymphocytic inflammation, which may be prevalent at the sebaceous gland level around the mid-follicle (Wilson, Burge, Dean, & Dawber, 1992). • *Histopathological diagnosis*: It is not always useful as the type of inflammatory infiltrate might be impossible to assess. In these cases, additional studies such as direct immunofluorescence can be useful (Villablanca et al., 2017). • *Trichoscopy*: A helpful tool in diagnosing the type of PSA and differentiating it from other types of alopecia (Oakley, 2015). Desquamation of the inner root sheath is a common presentation (Patterson, 2014). • *Laboratory testing*: It may be helpful to assess general patient health, immunology, and genetic links.	
Management	• The goal of treatment is to halt disease progress, prevent further hair loss, and reduce psychological impact (Patterson, 2014). • *Lifestyle*: The patient should avoid excessive heat exposure to the scalp, tight weaves, and braids (Patterson, 2014). • *Acute neutrophilic PSA*: Patients should be treated with a combination therapy incorporating systemic antibiotics (e.g., tetracycline, azithromycin), systemic corticosteroids, and isotretinoin. Review treatment effect at 2–6 months. Maintenance therapy should include a combination of topical antibiotics, corticosteroids, and 5% minoxidil (Villablanca et al., 2017). • *Acute lymphocytic PSA*: Management should combine systemic corticosteroids with antimalarials (i.e., hydroxychloroquine). Review treatment effect at 2–6 months. Patient maintenance should incorporate topical corticosteroids with minoxidil 5% (Villablanca et al., 2017). • *Recurrence*: SA has a high recurrence rate and can return with treatment cessation (Kolivras & Thompson, 2016; Villablanca et al., 2017). • *Hair transplant*: This is a challenge due to the extent of follicular damage and reduced vascularity of the scalp. Plus, for maximum success, the disease must be "burned out." • One study combined injections of platelet-rich plasma with follicular extraction and transplantation with excellent results (Saxena, Saxena, & Savant, 2016). • Another study showed success using a punch hair grafting technique in patients with end-stage CCCA (Davis & Callender, 2014). • *Prosthetics*: Wigs and hair pieces are an option and should be discussed in depth with patients who express or display anxiety or depression about their hair loss (Patterson, 2014). • *Support*: Refer the patient to local and online support groups as appropriate.	

Note. CCCA = central centrifugal cicatricial alopecia; PSA = primary scarring alopecia; SA = scarring alopecia; SSA = secondary scarring alopecia.

pathological findings may allow for a precise diagnosis in some cases; however, certainty is often difficult to achieve and reflects the limits of current dermatological knowledge of these disorders (Villablanca et al., 2017). Biopsy with histopathology and various hair tests are helpful in differentiating during the diagnostic process. Moving forward, the classification of alopecia will continue to evolve and change, with diagnostic clarity based primarily on trichoscopy findings (Kolivras & Thompson, 2016). As successful treatment of hair loss disorders in many cases

TABLE 5 Histopathology and Trichoscopy of Hair Loss Disorders

	Androgenetic Alopecia	Telogen Effluvium	Acute Alopecia Areata	Scarring Alopecia
Histopathology	 From *Androgenetic Alopecia [Dermatopathology/Dermatology/Histopathology Library Reference]*, by F. F. Soeprono, 2012a. Retrieved from http://www.dxpath.com/histlib/androgenetic-alopecia-histopathology-20497.html. Copyright 2012 by Fred F. Soeprono, MD. Reprinted with permission.	 From *Telogen Effluvium [Dermatopathology/Dermatology/Histopathology Library Reference]*, by F. F. Soeprono, 2012b. Retrieved from http://www.dxpath.com/histlib/telogen-effluvium-histopathology-20498.html. Copyright 2012 by Fred F. Soeprono, MD. Reprinted with permission.	 "Swarm of bees" around terminal hair follicles. From "Alopecia Areata: A Review," by S. S. Amin and S. Sachdeva, 2013, *Journal of the Saudi Society of Dermatology & Dermatologic Surgery, 17*(2), pp. 37–45. Copyright 2013 by Wolters Kluwer. Used with permission.	 Hyalinization of dermal collagen with broad fibrous tracts. From "Central Centrifugal Cicatricial Alopecia," by C. Blattner, D. C. Polley, F. Ferritto, and D. M. Elston, 2013, *Indian Dermatology Online Journal, 4*(1), pp. 50–51. Copyright 2013 by the Indian Dermatology Online Journal. All rights reserved. Reprinted with permission.
Trischoscopy	• Vellous hairs • Diversity in hair shaft thickness • Yellow dots (sebaceous debris) • Perifollicular pigmentation • Peripalar halo (Jain et al., 2013; Xu et al., 2017)	• Empty follicles • No hair shaft diameter diversity • No peripalar halo (Jain et al., 2013).	• Yellow dots (keratinous) • Exclamation mark hairs • Black dots (fractured dystrophic and telogen hairs) (Jain et al., 2013; Xu et al., 2017).	• Yellow dots and thick, arborizing vessels (lupus erythematous) • White dots (lichen planopilaris) • Scarred hypopigmented areas with follicular paucity • Follicular scaling and inflammation (folliculitis decalvans and dissecting folliculitis) (Jain et al., 2013; Xu et al., 2017)

is specific to the underlying disease type, it is important for clinicians to use currently available diagnostic tools and stay abreast of fresh, evidence-based approaches.

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