We are IntechOpen,
the world's leading publisher of
Open Access books
Built by scientists, for scientists

6,900
Open access books available

185,000
International authors and editors

200M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS
BOOK CITATION INDEX
INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Trichotillomania and Traction Alopecia

Tugba Kevser Uzuncakmak, Melek Aslan Kayıran,

Burak Tekin and Filiz Cebeci

Additional information is available at the end of the chapter

Abstract

Trichotillomania and traction alopecia are chronic habitual disorders characterized by repetitive pulling of hair that results in alopecia. They are commonly observed in children and adolescents but may present in adults due to occupational or traditional behavioral patterns. Trichotillomania (hair-pulling disorder) has been described more than a century ago, but we still have very limited data about its etiology and treatment. It is classified under the obsessive-compulsive and related disorders along with hoarding disorder, skin-picking disorder (excoriation) and body dysmorphic disorder in the *Diagnostic and Statistical Manual of Mental Disorder*s, Fifth Edition (DSM-5; American Psychiatric Association, May 2013). Traction alopecia is defined as loss of hair caused by repetitive or continuous and prolonged tension applied to the hair, usually on the scalp periphery and associated with mechanical traction of hair due to occupational behavioral patterns such as ballerinas or traditional behavioral patterns of hairstyles that cause tension. We aim to overview the clinical and diagnostic features of trichotillomania and traction alopecia and review the therapeutic options of these disorders in this chapter.

Keywords: alopecia, hair loss, psychosomatic disorders, traction alopecia, trichotillomania

1. Trichotillomania

1.1. Introduction

Trichotillomania is the disorder of repetitively pulling out one's hair from different areas of the body that results in noticeable hair loss [1]. The name trichotillomania was given to this behavior by a French dermatologist, Francois Henri Hallopeau, in 1889; however, the disorder also appears

in the literature in the works of Hippocrates. It is even found in plays by William Shakespeare such as Romeo and Juliet and The Life and Death of King John. The name is derived from the combination of Greek *thrix* for hair; *tillein*, plucking; and *mania*, madness.

Trichotillomania is classified under the obsessive-compulsive and related disorders along with hoarding disorder, skin-picking disorder (excoriation) and body dysmorphic disorder in the *Diagnostic and Statistical Manual of Mental Disorders* Fifth Edition (DSM-5; American Psychiatric Association, May 2013) [2] (**Table 1**). It is also grouped under obsessive-compulsive disorders in the eleventh revision of the World Health Organization's International Classification of Diseases and Related Health Problems (ICD-11) [3].

• Recurrent pulling out of one's hair, resulting in hair loss

• Repeated attempts to decrease or stop the pulling out of hair

• The hair pulling causes clinically significant distress or impairment in social, occupational, or other important areas of functioning

• The hair pulling or hair loss is not attributable to another medical condition

• The hair pulling is not better explained by the symptoms of another mental disorder

---

Table 1. Diagnostic criteria (DSM-5) [2].

1.2. Epidemiology

Trichotillomania occurs more frequently in females. The lifetime prevalence is 3.4% for women and 1.5% for men [4]. The typical age of onset is between 5 and 12 years or early childhood to adolescence, but it may occur in any age.

1.3. Pathogenesis

The onset of trichotillomania often occurs after a stressful event such as the divorce of parents, loss of a loved one or unemployment [5]. Occasionally, trichotillomania is only seen while sleeping, a condition known as sleep-isolated trichotillomania [6].

1.4. Diagnosis

1.4.1. Clinical features

Patients may have other problems with self-mutilation such as nail-biting or dermatitis artefacta. Approximately one-third of the patients chew or swallow the hair they pull out, which is called trichophagia and some of them develop trichobezoars, which is the accumulation of the patients' own hair in the intestines. These trichobezoars may result in a "tail" that lies along the duodenum, a phenomenon which is called *Rapunzel Syndrome*. Around 1% of trichobezoar patients may need surgical intervention [4].

Symptom severity can be measured by using different validated instruments including Massachusetts General Hospital Hair Pulling Scale that has seven parameters, rating symptom

severity from 0 to 4 and assessing various aspects of plucking during the past seven days: actual pulling, urge to pull, associated distress and perceived control, The Yale-Brown Obsessive-Compulsive Scale, The Psychiatric Institute Trichotillomania Scale, The Trichotillomania Scale for Children, The Milwaukee Inventory for Styles of Trichotillomania-Child Version [7]. The MGHHS includes seven parameters, rating symptom severity from 0 to 4 and assesses several aspects of hair pulling during the previous 7 days: urge to pull, actual pulling, perceived control and associated distress. The MGHHS and its Dutch adaptation have been reported to provide good psychometric properties [7].

Three subtypes of hair pulling have been described (**Table 2**).

• Early onset: occurring in young children, mostly under the age of 8; usually does not need any treatment.

• Automatic: occurring when the individual is busy with other activities, such as reading. This type affects 75% of patients.

• Focused: occurs with the patient's attention and is associated with strong impulses to pull hair.

Table 2. Subtypes of trichotillomania.

Any part of the hair may be affected, but the targeted hair is mostly on the scalp (75%). The eyelashes (53%), eyebrows (42%), pubic region (17%) and beard (10%) may also be involved, but sometimes there is more than one location (17%) [8, 9]. The most affected scalp areas are the frontoparietal region and vertex, while the least affected region is the occiput. The lower eyelid is usually not involved; this is helpful to distinguish trichotillomania from alopecia areata [10].

Alopecic plaques are usually located on the contralateral side of the dominant hand. There may be more than one plaque. These plaques of hair loss most often have irregular shapes and contain many broken hairs of varying lengths. The margins have normal and long hair.

The plucked hairs have fiber fractures and feel rough on examination. There are usually no signs of inflammation in the plaques, but there may also be signs of excoriation, lichenification and post-inflammatory hyperpigmentation in some cases.

1.4.2. Dermoscopy

Trichoscopy may also be utilized for differential diagnosis. The trichotillomania plaque includes broken and irregular coiled hair and hair density is decreased. Black dots, follicular hemorrhages and V-sign may be seen [11]. Trichoscopy is very useful in the differential diagnosis of trichotillomania from alopecia areata where exclamation mark hairs, yellow dots and proximal tapering hairs could be seen, in contrast with trichotillomania where trichoptilosis, pointed hairs, flame hairs, V-sign, hook hairs, hair powder, follicular microhemorrhage and tulip hairs are more characteristic. Follicular microhemorrhage meaning a red dot that corresponds to a follicular ostium stuffed with blood clot, may support local trauma, a clue for trichotillomania [12].

1.4.3. Histopathology

The diagnosis of trichotillomania is usually made by clinical examination and patient history. However, occasionally especially in pediatric patient group both child and parents may deny the possibility of pulling or plucking as a cause of hair loss and especially in localized involved patients the diagnosis may be difficult. Histopathological examination may be necessary in these patients. Follicules of normal size, increased catagen and telogen hairs (up to 75%) which is a result of mechanical trauma to the hair frequently propelling anagen follicules into the catagen phase, pigmentary defects and casts, evidence of traumatized hair bulbs and tricho-malacia (a complete but distorted, fully developed terminal hair in its bulb) are the most common findings in histological examination of trichotillomania [13, 14]. Catagen hairs may be present in areas that have recently been injured and telogen hairs may present after a few weeks from pulling. Some hair follicles in anagen phase may be present, but they are usually seen empty because of hair shaft avulsion. If the hair matrix and suprabulbar epithelium are injured, but not severely disrupted, the follicle may remain in the anagen phase which may produce a hair shaft. Follicles can show distortion of the bulbar epithelium and sometimes conspicuous hemorrhage.

Hair shaft avulsion may deposit melanin pigment in the hair papilla and peribulbar connective tissue [13, 14]. Frequently, chunks of pigmented hair matrix or cortex cells are torn from their moorings during the plucking process and come to rest in superficial portions of the follicles. These cells then shrink to form a dark black homogeneous clump called a pigment cast. Pigment casts which are very characteristic for trichotillomania simply occur as the by-product of fragmented, ectopic matrix or cortical epithelium and usually seen in the isthmus or infundibulum. Trichomalacia, which defines shaft abnormalities such as distorted in shape, smaller than normal and incompletely cornified, is very characteristic for trichotillomania.

These injuries of pulling or plucking to the bulbar portions of follicles do not induce inflammation but may cause follicular microhemorrhage within the lower portion of the follicle. A few eosinophils may be rarely seen around the lower portion of the traumatized follicule. Also miniaturization of follicules is usually not seen in trichotillomania and absence of inflammatory infiltrate and loss of miniaturization are usually serve to differentiate it from alopecia areata [14].

1.5. Differential diagnosis

Tinea capitis, alopecia areata, loose anagen hair, monilethrix, lichen planopilaris and secondary syphilis need to be considered in the differential diagnosis of trichotillomania. Trichoscopy, medical history and scalp biopsy can be used to distinguish trichotillomania from other diseases. Catagen and telogen hair numbers are found to be increased and usually there are no signs of inflammation unless there is an infection in histologic examination. The number of catagen hairs exceeds telogen hairs in chronic lesions. Perifollicular hemorrhage may be found at the circumference of the hair bumbs [1].

Potassium hydroxide examination, fungal culture and Wood's lamp examination may be performed to exclude tinea capitis. The hair is weak and may easily be pulled out in tinea capitis.

Alopecia areata plaques are oval and well-demarked. A hair pull test may be helpful as a diagnostic test for alopecia areata. Telogenic hairs may be pulled out easily in alopecia areata, which indicates the activity of the disease in contrast with trichotillomania. Shaving the involved area and waiting for the regrowth may also be useful for the diagnosis of trichotillomania. Alternatively, a small part of the hair is clipped near the scalp with scissors and the hairs in trichotillomania display uniform hair regrowth.

Trichoscopy may also be utilized for differential diagnosis. Exclamation mark hairs and yellow dots may be seen and white hairs are usually not involved in alopecia areata [11]. The trichotillomania plaque has broken and irregular coiled hair and the hair density is decreased. Black dots, follicular hemorrhages, v-sign may be seen [12].

1.6. Treatment

As mentioned above, trichotillomania is a psychiatric disorder with dermatological findings which is characterized by compulsive avulsion of hair shafts leading to thin, ragged, broken hairs on the affected region clinically [15]. The inability to control self-pulling of hair resulting in hair loss may progress into alopecia in long time.

Treatment procedure must be multidisciplinary including both dermatological but mainly psychiatric approach to increase the effectiveness of the therapy and prevent relapse.

Cognitive and behavioral therapies (CBT), antipsychotic agents, selective serotonin reuptake inhibitors (SSRI), tricyclic antidepressants are the main options of treatment [7, 16–18]. The most appropiate therapeutic approach must be chosen according to the patient's age, medical status and mental status.

Cognitive and behavioral therapies are the first steps of treatment and must be considered together with pharmacotherapeutics in treatment [16, 19]. In a randomized controlled trial with 7–8-year-olds, cognitive-behavioral therapies alone were found to decrease the symptoms in 75% of the participants [16].

The results about efficacy of SSRIs are conflicting. They were reported to be the safest and well-established medication choice. However, the clinical results show that medication, which is usually an SSRI, in addition to CBT, is more successful, if CBT alone fails.

In a meta-analysis which was reported in 2007, in which the efficacies of pharmacologic and behavioral treatments were evaluated in treatment of trichotillomania, it was found that SSRIs were not more effective than placebo. In two trials clomipramine was found to be more effective compared with placebo. In three trials, it was shown that there is a beneficial effect of habit reversal therapy compared with no intervention [20].

In another systematic review which was published in 2013, similar results were found. In two included trials, fluoxetine was not more effective than placebo in reducing the mean severity

rating of hair pulling. Clomipramine was found to be more effective than placebo, although 3 of 10 participants receiving clomipramine dropped out because of drug-related adverse effects [21].

At last, in a recent meta-analysis of 11 randomized trials, the efficacy of behavioral therapy and SSRI for the treatment of trichotillomania; the outcome measure was the standardized mean difference of change in hair pulling [22]. This publication demonstrated a large effect for behavioral therapy and only a moderate effect for SSRI. A greater treatment effect was reported for clomipramine in two included trials [22].

The side effects and limited efficacy of pharmacological treatment, especially in pediatric population and difficulty in long-term maintenance of behavioral therapies require alternative options of treatment. The glutamatergic system dysregulation is involved in obsessive-compulsive disorders etiology and it has been reported that N-acetyl-cysteine (NAC) might have a therapeutic effect on these entities by acting on the glutamatergic system and reducing oxidative stress [23]. It was reported as a safe and effective treatment option given 1200 mg/d per os. The efficacy of the glutamate modulator NAC was evaluated in a small randomized trial including 50 adults with trichotillomania [24]. N-acetylcysteine was more effective than placebo in reducing hair-pulling symptoms as measured by the Massachusetts General Hospital Hair Pulling Scale. A subsequent trial in children and adolescents did not find any beneficial effect of NAC compared with placebo [24, 25].

2. Traction alopecia

2.1. Introduction

Traction alopecia (TA) is defined as loss of hair caused by repetitive or continuous and prolonged tension applied to the hair [26–28]. It was first described using the terminology *alopecia groenlandica* to refer to the hair loss attributed to tight ponytails which girls and women wear in Greenland [29]. Although TA is more prevalent among females of African ancestry, it has been described in a wide range of populations, including nurses, ballerinas, Sikh boys and men, to name a few [30–34]. Research has convincingly demonstrated that certain habitual practices of hairstyling are implicated in the pathogenesis of TA [35–39].

2.2. Epidemiology

Most of the population-based studies concerning the prevalence of TA originate from South Africa [35–37]. Overall, TA was found to be more common in females compared to males and in women compared to girls, presumably due to a longer history of hairdressing [37]. Importantly, in a large cohort of African adults in Cape Town, almost one-third of women had findings consistent with TA on scalp examination [35]. Of note, 17.1% of South African schoolgirls had TA, with increasing prevalence rates from the first year of school to the last year of high school [36]. A similar rate (18.4%) was observed in another study examining a population of 201 African American girls from the United States [38]. In males, TA seems to

be considerably less prevalent, however, two notable exceptions are Sikh boys and men, who adhere to the religious practice of tightly knotting their scalp and/or beard hair and boys/men wearing dreadlocks or cornrows [32–35, 40].

2.3. Pathogenesis

Traction alopecia is typically noncicatricial and reversible in its early stages, whereas it may progressively result in permanent scarring in the long term [27, 28, 41]. It is well recognized that traction causes an inflammatory, sometimes subclinical, folliculitis. The exact pathomechanism leading from follicular inflammation to follicle damage and hair loss remains to be elucidated; however, follicular miniaturization is considered to play a possible role [28, 42–44].

2.4. Predisposing factors

The factors predisposing to the development of TA fall into two major categories: (1) traumatic hairstyling practices and (2) application of chemicals and/or heat to the hair [27]. It has been shown that the combination of both factors greatly increases the risk of TA, cautioning against the application of traction on chemically relaxed hair [36–38]. Importantly, hairdressing practices causing symptoms such as stinging, pain, or crusting are associated with an increased risk of TA [37, 39]. "Tenting" of the hair follicle, which manifests as elevation of the scalp skin due to tight pulling, has been interpreted as a sign of excessive tension [27, 39, 43]. A more recent literature review classified hairstyles into low risk, moderate risk and high-risk [39]. Accordingly, the excessive use of very tight buns or ponytails belongs to the category of high-risk hairstyles, as do dreadlocks, cornrows and braids [38, 39, 45]. Another well-recognized factor associated with TA is the use of hair extensions, especially if applied to relaxed hair [39, 46]. According to the aforementioned review, other hairstyles such as braids and/or weaves are considered high-risk if they are combined with chemical relaxation of the hair [39]. Moreover, hairpins used to fix the nurse's cap to the scalp may be related to hair loss [30]. Alopecia initially presenting with a traumatic ulcer was described in association with hairstyles requiring multiple hairpins [47].

2.5. Diagnosis

2.5.1. Clinical findings

Two main categories of TA have been recognized: marginal and nonmarginal [46]. The former is more common and typically presents as bandlike loss of hair along the temporoparietal margin or frontal hairline (**Figure 1**) [48]. Marginal TA is usually attributable to traumatic hairstyles, whereas nonmarginal TA may be caused by hairpins or buns [40, 47, 49]. A peculiar form of TA, termed "horseshoe pattern" by the authors, was described as a result of weft hair extensions [46]. Hair loss in nonmarginal localizations may sometimes present a diagnostic challenge and require more detailed history taking and/or histopathological examination [27, 48].

Figure 1. Marginal traction alopecia along the frontal hairline **(A)** and temporoparietal margin **(B)** in a woman who applied excessive traction to her hair.

The earliest clinical sign of TA is considered to be perifollicular erythema in the areas of the scalp exposed to maximum tension, which may progress to folliculocentric papules and pustules. However, these initial findings may be unrecognized by the patient and physician alike [22, 28, 31]. The presentation may be acute or more commonly, chronic and progressive. Patients may provide a history of symptoms such as stinging or tenderness during hairdressing practices [27]. As noted earlier, the alopecia is typically nonscarring in the early stages, but may become irreversible later during the disease course, following a "biphasic" course [27]. Correspondingly, follicular markings tend to be decreased in the late stages of TA [27, 31, 43]. An important clinical caveat in the diagnosis of TA is the "fringe sign," defined as the presence of retained hairs along the frontal and/or temporal margin. This useful finding was observed in early and late stages of TA alike (**Figure 2**) [43].

In general, TA is considered to have no systemic associations. Nonetheless, there is an anecdotal report of a 25-year-old woman with prolonged traction resulting in a combination of TA, cutis verticis gyrata and intractable headache. Her headache resolved as she was advised to change her habitual hairstyle, which also stopped further hair loss [50].
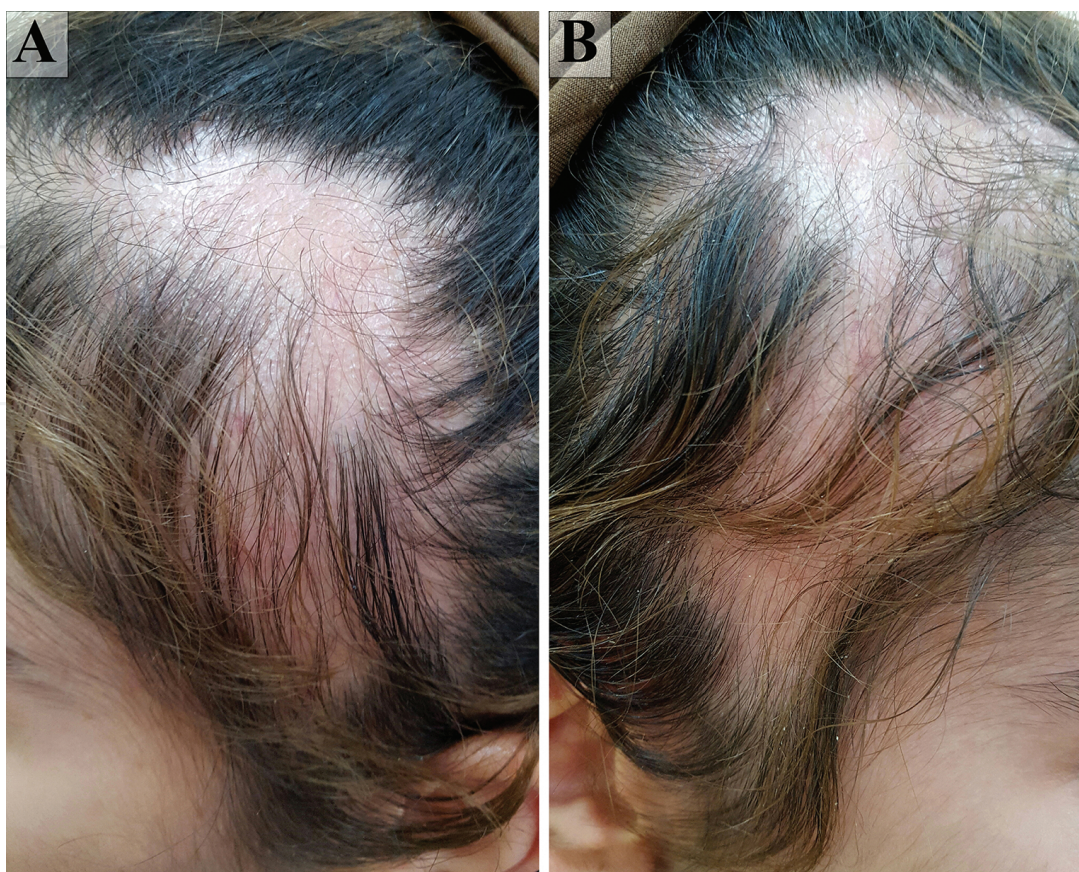
Figure 2. "Fringe sign" demonstrated in a woman with longstanding traction alopecia as a group of retained hairs along the temporoparietal hairlines and located in front of the alopecic patches bilaterally.

2.5.2. Dermoscopy

Dermoscopy may be utilized as a useful aid to confirm the clinical diagnosis of TA and/or differentiate it from other entities presenting with hair loss. In a cross-sectional study from Korea, broken hairs and black dots were observed in 100 and 92% of patients with TA, respectively. Of these findings, broken hairs were noted in all patients with trichotillomania, as well (**Figure 3**). Other dermoscopic findings associated with TA in this study were clustered short vellus hairs, yellow dots and atypical red vessels, with decreasing frequency [51]. The presence of hair casts has been emphasized as an important dermoscopic sign of TA, described as cylindrical structures encircling the proximal hair shafts [52]. It has been demonstrated that observation of hair casts on dermoscopic examination is an indicator of ongoing traction [53] and should be interpreted as a warning sign that hairstyle changes should be implemented to halt the progression of alopecia [52]. More recently, the trichoscopic finding of "flame hairs"was reviewed in a population of patients with various hair disorders. Of note, flame hairs were noted in slightly more than one half of the patients with trichotillomania, whereas they were observed in less than 5% of patients with TA. This disparity was attributed by the authors to the extent of the acute mechanical damage to the hair follicle being more prominent in trichotillomania compared to that in TA [54].

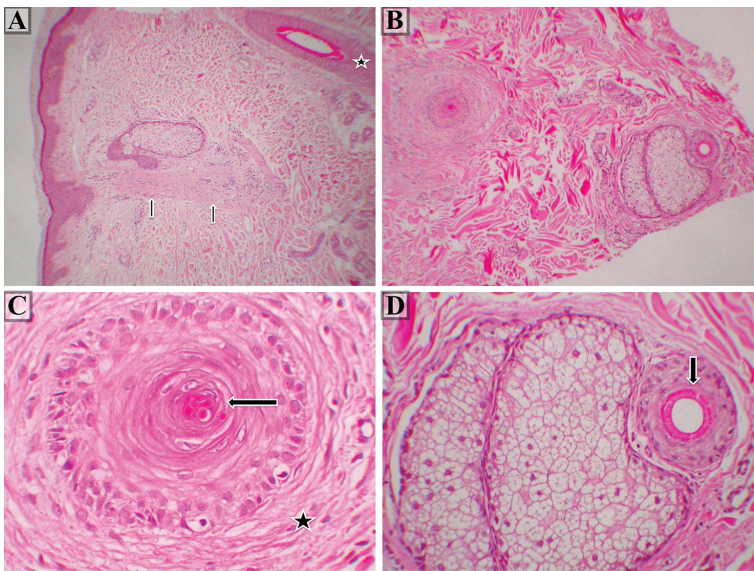**Figure 3.** Broken and irregular coiled hairs, decreased hair density in dermoscopic examination.

Figure 4. (A) Perifollicular fibrosis on superficial dermis ◀▬ and an anagen follicule with hair shaft avulsion ★- H&Ex100, **(B)** catagen/telogen hair follicules- H&Ex40, **(C)** intrafollicular erythrocytes ◀▬ and perifollicular fibrosis ★ H&Ex400 and **(D)** an empty follicule with hair shaft avulsion ◀▬ H&E ×200.

2.5.3. Histopathology

Histologic findings of TA parallel the biphasic course of the disease mentioned previously [27]. Early stages of TA are considered to resemble trichotillomania, whereas more advanced stages of TA are similar to "burned-out" forms of cicatricial alopecia [55]. More specifically, early TA is characterized by increased numbers of telogen and catagen hairs and trichomalacia. In contrast to primary scarring alopecias, sebaceous glands are generally preserved in TA (**Figure 4**). With prolonged traction, terminal follicles tend to decrease in number and are progressively replaced by fibrous tracts [27, 28, 43]. Furthermore, vellus-sized hairs may be

observed on histology which are thought to correspond to the aforementioned "fringe sign" noted on physical examination [27]. Differentiation of late-stage TA from primary scarring alopecias may be challenging and it has been demonstrated that transverse sections may be advantageous compared to vertical sections in differential diagnosis [56].

2.6. Differential diagnosis

The clinical differential diagnosis of TA depends on the distribution pattern of hair loss. For marginal type TA, the most important entities to be considered in the differential diagnosis are alopecia areata with an ophiasis pattern and frontal fibrosing alopecia [27, 43]. A noteworthy caveat is that TA only affects scalp hair exposed to traction, whereas body hair, eyebrows and/or nails may be involved in alopecia areata or frontal fibrosing alopecia [27]. Another important differential diagnosis is androgenetic alopecia. These two conditions may also coexist and in fact androgenetic alopecia is thought to predispose an individual to the development of TA due to the miniaturized hairs [30, 40, 43]. Nonmarginal TA, on the other hand, may be considered within the differential diagnosis of a broad range of conditions, including alopecia areata, trichotillomania, telogen effluvium and discoid lupus erythematosus [40, 48].

Central centrifugal cicatricial alopecia (CCCA) is another condition mainly seen in females of African descent [57]. The relationship between traction/TA and CCCA remains controversial [58]. Ackerman and coauthors categorized CCCA as a form of TA, [59] and a retrospective comparative study detected a strong association between CCCA and the use of tractional hairstyles with artificial hair extensions [60]. However, a more recent study failed to reveal an association between CCCA and tractional hairstyles. Interestingly, among more than 1000 female participants, not a single individual had a concomitant diagnosis of CCCA and TA in the same study, suggesting that CCCA and TA may not be closely related [58].

2.7. Prevention and treatment

Prevention is an integral part of management of TA, as appropriate measures to eliminate traction can stop hair loss before it becomes permanent [27, 28, 61]. It is important to recognize that TA most often originates during childhood and adolescence, suggesting that public education should primarily focus on these at-risk populations [27, 43, 44]. Prevention strategies have been detailed elsewhere [39, 44]. Briefly, management should rely on two main principles: preferring loose hairstyles and avoiding heat and chemicals [27, 39]. Patients may be encouraged to switch from high-risk hairstyles to low-risk hairstyles, such as loose buns and ponytails [39]. An important message is that pain during hairstyling be interpreted as a sign that excessive tension has been applied and that particular hairstyle should better be avoided [39, 44].

Medical or surgical treatment should only be considered after traction has been minimized [39]. There is a paucity of evidence-based literature data with regard to the medical treatment of TA. Topical or intralesional corticosteroids and topical or systemic antibiotics may be utilized to suppress the inflammation in the early stages of the disease [61].Topical min-

oxidil was described as effective in an anecdotal report [42] and recommended by some authors [27, 43, 61], although controlled trials are lacking. An interesting study demonstrated the protective effect of piloerection induced by topical phenylephrine against the development of TA, suggesting a potential role of α_1-adrenergic receptor agonists in the treatment of TA [41]. For extensive TA, surgical treatment has been described [45, 62]. Of note, a scoring system was developed ("M-TAS score") to evaluate the severity of marginal type TA and facilitate assessment of treatment effectiveness [63].

Author details

Tugba Kevser Uzuncakmak[*], Melek Aslan Kayıran, Burak Tekin and Filiz Cebeci

*Address all correspondence to: drtugbakevser@gmail.com

Department of Dermatology, Medical Faculty, Goztepe Training and Research Hospital, Istanbul Medeniyet University Istanbul, Turkey

References

[1] Zimova J, Zimova P. Trichotillomania: bizarre pattern of hair loss at 11-year-old girl. Acta Dermatovenerol Croat. 2016; 24: 150–153.

[2] Woods DW, Houghton DC. Diagnosis, evaluation and management of trichotillomania. Psychiatry Clin North Am. 2014; 37: 301–317.

[3] Stein DJ, Kogan CS, Atmaca M, Fineberg NA, Fontenelle LF, Grant JE, Matsunaga H, Reddy YC, Simpson HB, Thomsen PH, van den Heuvel OA, Veale D, Woods DW, Reed GM. The classification of obsessive-compulsive and related disorders in the ICD-11. J Affect Disord. 2016; 190: 663–674. DOI: 10.1016/j.jad.2015.10.061.

[4] Yik YI, How AK. A 'Hairy' problem: trichotillomania, trichophagia and trichobezoars. Singapore Med J. 2016 Jul;57(7):411. DOI: 10.11622/smedj.2016125.

[5] Nisha SC, Jeroen N, Matilde I, Ramon G and Arnold PO. Trichotillomania in children. Skin Appendage Disord. 2015; 1: 18–24.

[6] Goyal D, Surya S, Elder J, McCall WV, Graham K. Sleep-isolated trichotillomania (SITTM): a case report. Innov Clin Neurosci. 2016; 13: 40–42.

[7] Keijsers GP, Maas J, van Opdorp A, van Minnen A. Addressing self-control cognitions in the treatment of trichotillomania: a randomized controlled trial comparing cognitive therapy to behaviour therapy. Cognit Ther Res. 2016; 40: 522–531.

[8] Carli Murphy, B.S., Rachel Redenius, Erin O'Neill, Sarah Zallek, M.D. Sleep-isolated trichotillomania: a survey of dermatologists. J Clin Sleep Med. 2007; 3: 719–721.

[9] Shoenfeld N, Rosenberg O, Kotler M, Dannon PN.Tricotillomania: pathopsychology theories and treatment possibilities. Isr Med Assoc J. 2012 Feb;14(2):125–9.

[10] Grillo E, Vano-Galvan S, Diaz-Ley B, Jaén P Patchy hair loss on the pubis—a case study. Aust Fam Physician. 2013; 42: 487–489.

[11] Finner AM Alopecia areata: Clinical presentation, diagnosis and unusual cases. Dermatol Ther. 2011; 24: 348–354. DOI: 10.1111/j.1529-8019.2011.01413.

[12] Ankad BS, Naidu MV, Beergouder SL, Sujana L. Trichoscopy in trichotillomania: a useful diagnostic tool. Int J Trichol. 2014 Oct;6(4):160–163. DOI: 10.4103/0974-7753.142856

[13] Muller SA. Trichotillomania: a histopathologic study in sixty-six patients. J Am Acad Dermatol. 1990; 23: 56–62.

[14] Elder DE, Elenitsas R, Johnson BL, Ioffreda MD, Miller J, Miller OF. Inflammatory and other benign disorders of skin appendages. In: Atlas and Synopsis of Lever's Histopathology of the Skin, 2nd Edition. China: Lippincott Williams & Wilkins, 2007. pp. 408–410.

[15] Basta-Juzbašić A, Bukvić Mokos Z. Skin changes in primary psychiatric disorders. Acta Dermatovenerol Croat. 2015; 23: 87–95.

[16] Landuyt G, Dierckx B, de Nijs PF, Dieleman GC. Treatment options for paediatric trichotillomania. Tijdschr Psychiatr. 2016; 58: 463–470.

[17] Sasaki T, Iyo M. Treatment of puberty trichotillomania with low-dose aripiprazole. Ann Gen Psychiatry. 2015;14: 18. DOI: 10,1186/s12991-015-0056-0.

[18] Rothbart R, Stein DJ. Pharmacotherapy of trichotillomania (hair pulling disorder) an updated systematic review. Expert Opin Pharmacother. 2014; 15: 2709–2719. DOI: 10.1517/14656566.2014.972936.

[19] Trainor K. Treating trichotillomania in children and adolescents: CBT versus medication. Am J Psychiatry. 2007; 164: 1610–1611.

[20] Bloch MH, Landeros-Weisenberger A, Dombrowski P, et al. Systematic review: pharmacological and behavioral treatment for trichotillomania. Biol Psychiatry 2007; 62: 839.

[21] Ninan PT, Rothbaum BO, Marsteller FA, et al. A placebo-controlled trial of cognitive-behavioral therapy and clomipramine in trichotillomania. J Clin Psychiatry 2000; 61: 47.

[22] McGuire JF, Ung D, Selles RR, et al. Treating trichotillomania: a meta-analysis of treatment effects and moderators for behavior therapy and serotonin reuptake inhibitors. J Psychiatr Res 2014; 58: 76.

[23] Grant JE, Odlaug BL, Kim SW. N-acetylcysteine, a glutamate modulator, in the treatment of trichotillomania: a double-blind, placebo-controlled study. Arch Gen Psychiatry 2009; 66: 756.

[24] Bloch MH, Panza KE, Grant JE, et al. N-Acetylcysteine in the treatment of pediatric trichotillomania: a randomized, double-blind, placebo-controlled add-on trial. J Am Acad Child Adolesc Psychiatry 2013; 52: 231.

[25] Smith L, Tracy DK, Giaroli G. What future role might N-acetyl-cysteine have in the treatment of obsessive compulsive and grooming disorders?: A systematic review. J Clin Psychopharmacol. 2016; 36: 57–62. DOI: 10.1097/JCP.0000000000000431.

[26] Whiting DA. Traumatic alopecia. Int J Dermatol. 1999; 38:34–44.

[27] Mirmirani P, Khumalo NP. Traction alopecia: how to translate study data for public education—closing the KAP gap? Dermatol Clin. 2014; 32:153–161. DOI: 10.1016/j.det. 2013.12.003

[28] Khumalo NP, Callender VD. African hair. In: Blume-Peytavi U, Tosti A, Whiting DA, Trüeb RM, editors. Hair Growth and Disorders. 1st ed. Berlin: Springer; 2008. p. 483–507. DOI: 10.1007/978-3-540-46911-7

[29] Hjorth N. Traumatic marginal alopecia; a special type: alopecia groenlandica. Br J Dermatol. 1957; 69:319–322.

[30] Hwang SM, Lee WS, Choi EH, Lee SH, Ahn SK. Nurse's cap alopecia. Int J Dermatol. 1999; 38:187–191.

[31] Samrao A, Chen C, Zedek D, Price VH. Traction alopecia in a ballerina: clinicopathologic features. Arch Dermatol. 2010;146:930–931. DOI: 10.1001/archdermatol.2010.183.

[32] Kanwar AJ, Kaur S, Basak P, Sharma R. Traction alopecia in Sikh males. Arch Dermatol. 1989; 125:1587.

[33] James J, Saladi RN, Fox JL. Traction alopecia in Sikh male patients. J Am Board Fam. Med 2007; 20:497–498.

[34] Karimian-Teherani D, El Shabrawi-Caelen L, Tanew A. Traction alopecia in two adolescent Sikh brothers-an underrecognized problem unmasked by migration. Pediatr Dermatol. 2011; 28:336–338. DOI: 10.1111/j.1525-1470.2011.01241.x

[35] Khumalo NP, Jessop S, Gumedze F, Ehrlich R. Hairdressing and the prevalence of scalp disease in African adults. Br J Dermatol. 2007; 157:981–988. DOI: 10.1111/j.1365-2133.2007.08146.x

[36] Khumalo NP, Jessop S, Gumedze F, Ehrlich R. Hairdressing is associated with scalp disease in African schoolchildren. Br J Dermatol. 2007; 157:106–110. DOI: 10.1111/j.1365-2133.2007.07987.x

[37] Khumalo NP, Jessop S, Gumedze F, Ehrlich R. Determinants of marginal traction alopecia in African girls and women. J Am Acad Dermatol. 2008; 59:432–438. DOI: 10.1016/j.jaad.2008.05.036

[38] Rucker Wright D, Gathers R, Kapke A, Johnson D, Joseph CL. Hair care practices and their association with scalp and hair disorders in African American girls. J Am Acad Dermatol. 2011; 64:253–262. DOI: 10.1016/j.jaad.2010.05.037

[39] Haskin A, Aguh C. All hairstyles are not created equal: What the dermatologist needs to know about black hairstyling practices and the risk of traction alopecia (TA). J Am Acad Dermatol. DOI: 10.1016/j.jaad.2016.02.1162

[40] Hantash BM, Schwartz RA. Traction alopecia in children. Cutis. 2003; 71:18–20.

[41] Goren A, Shapiro J, Sinclair R, Kovacevic M, McCoy J. α1-AR agonist induced piloer-ection protects against the development of traction alopecia. Dermatol Ther. 2016; 29:160–163. DOI: 10.1111/dth.12324

[42] Khumalo NP, Ngwanya RM. Traction alopecia: 2% topical minoxidil shows promise. Report of two cases. J Eur Acad Dermatol Venereol. 2007; 21:433–434. DOI: 10.1111/j.1468-3083.2006.01933.x

[43] Samrao A, Price VH, Zedek D, Mirmirani P. The "Fringe Sign"—a useful clinical finding in traction alopecia of the marginal hair line. Dermatol Online J. 2011; 17:1.

[44] Khumalo NP. The "fringe sign" for public education on traction alopecia. Dermatol Online J. 2012; 18:16.

[45] Ozçelik D. Extensive traction alopecia attributable to ponytail hairstyle and its treat-ment with hair transplantation. Aesthetic Plast Surg. 2005; 29:325–327. DOI: 10.1007/s00266-005-0004-5

[46] Ahdout J, Mirmirani P. Weft hair extensions causing a distinctive horseshoe pattern of traction alopecia. J Am Acad Dermatol. 2012; 67:e294–295. DOI: 10.1016/j.jaad.2012.07.020

[47] Ntuen E, Stein SL. Hairpin-induced alopecia: case reports and a review of the literature. Cutis. 2010; 85:242–245.

[48] Barbosa AB, Donati A, Valente NS, Romiti R. Patchy traction alopecia mimicking areata. Int J Trichol. 2015; 7:184–186. DOI: 10.4103/0974-7753.171588

[49] Trüeb RM. "Chignon alopecia": a distinctive type of nonmarginal traction alopecia. Cutis. 1995; 55:178–179.

[50] Kanwar AJ, Ghosh S, Thami GP, Kaur S. Alopecia and cutis verticis gyrata due to traction presenting as headache. Int J Dermatol. 1992; 31:671–672.

[51] Shim WH, Jwa SW, Song M, Kim HS, Ko HC, Kim BS, Kim MB. Dermoscopic approach to a small round to oval hairless patch on the scalp. Ann Dermatol. 2014; 26:214–220. DOI: 10.5021/ad.2014.26.2.214.

[52] Tosti A, Miteva M, Torres F, Vincenzi C, Romanelli P. Hair casts are a dermoscopic clue for the diagnosis of traction alopecia. Br J Dermatol. 2010; 163:1353–1355. DOI: 10.1111/ j.1365-2133.2010.09979.x

[53] Zhang W. Epidemiological and aetiological studies on hair casts. Clin Exp Dermatol. 1995; 20:202–207.

[54] Miteva M, Tosti A. Flame hair. Skin Appendage Disord. 2015; 1:105–109. DOI: 10.1159/000438995

[55] Sperling LC. Scarring alopecia and the dermatopathologist. J Cutan Pathol. 2001; 28: 333–342.

[56] Donovan JC, Mirmirani P. Transversely sectioned biopsies in the diagnosis of end-stage traction alopecia. Dermatol Online J. 2013; 19:11.

[57] Whiting DA, Olsen EA. Central centrifugal cicatricial alopecia. Dermatol Ther. 2008; 21: 268–278. DOI: 10.1111/j.1529-8019.2008.00209.x

[58] Khumalo NP, Gumedze F. Traction: risk factor or coincidence in central centrifugal cicatricial alopecia? Br J Dermatol. 2012; 167: 1191–1193. DOI: 10.1111/j.1365-2133.2012.11050.x

[59] Ackerman AB, Charissi C, Jones RE, Walton NW III. Hot comb alopecia/follicular degeneration syndrome in African American women is traction alopecia! Dermatopathol Pract Concept. 2000; 6: 320–336.

[60] Gathers RC, Jankowski M, Eide M, Lim HW. Hair grooming practices and central centrifugal cicatricial alopecia. J Am Acad Dermatol. 2009; 60:574–578. DOI: 10.1016/ j.jaad.2008.10.064.

[61] Callender VD, McMichael AJ, Cohen GF. Medical and surgical therapies for alopecias in black women. Dermatol Ther. 2004; 17: 164–176. DOI: 10.1111/j.1396-0296.2004.04017.x

[62] Earles RM. Surgical correction of traumatic alopecia marginalis or traction alopecia in black women. J Dermatol Surg Oncol. 1986; 12: 78–82.

[63] Khumalo NP, Ngwanya RM, Jessop S, Gumedze F, Ehrlich R. Marginal traction alopecia severity score: development and test of reliability. J Cosmet Dermatol. 2007; 6:262–269. DOI: 10.1111/j.1473-2165.2007.00345.x