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Psychosomatic Dimension in Alopecia Areata: Literature Review illustrated with a Clinical Case.

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Artigo de Revisão Narrativa ilustrado com Caso Clínico

Dissertação de candidatura ao grau de Mestre em Medicina,
submetida ao Instituto de Ciências Biomédicas Abel Salazar da Universidade do Porto

Psychosomatic Dimension in Alopecia Areata: Literature Review illustrated with a Clinical Case.

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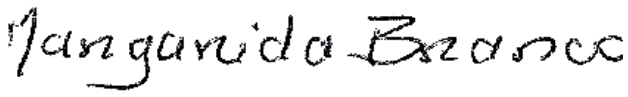
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Ao doente incluído neste estudo de caso, pela sua colaboração informada e voluntária na partilha de informação clínica e psicopatológica relevante, essencial para a concretização deste trabalho.

Intenção de Publicação

A seguinte revisão narrativa, ilustrada com caso clínico, encontra-se em fase precoce de maturação, tendo sido estruturada com a intenção de publicação numa revista científica na área de interesse.

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Abstract

Introduction and Objectives

The objective of this dissertation, illustrated with a clinical case, is to provide a narrative literature review on contemporary psychosomatic medicine, with a particular focus on the field of psychodermatology and a comprehensive understanding of alopecia areata. Additional aims include: informing healthcare professionals – namely dermatologists, psychiatrists, and psychologists – of the critical role played by patients' psychological functioning and, often times, underlying psychopathology in psychosomatic disorders, as well as the importance of interdisciplinary care; raising awareness among health education institutions of the need to incorporate concepts of psychosomatic medicine into medical training, given their relevance across a wide range of clinical presentations; and promoting, among policy-makers and healthcare administrators, the recognition of the impact of integrated approaches in the management of complex conditions, including improvements in quality of life, increased productivity, and reduced long-term healthcare costs.

Methodology

(1) A literature search was conducted using PubMed Central, SCOPUS, PsycINFO and PsycNET databases for articles published between 1 January 2015 and 1 June 2025. The following Medical Subject Headings (MeSH) terms were used as search keywords and combined in pairs: *"Alopecia Areata", "Alexithymia", "Multidisciplinary Care Team", "Psychodermatology", "Psychological Factors", "Psychopathology", "Psychosomatic Medicine"* and *"Therapeutics"*. In addition, other relevant articles, including source references of each selected article, were also included. All publications were screened by title, abstract, and full-text review and selection was made in accordance with the dissertation's objectives. Priority was given to original articles, systematic reviews, meta-analyses and peer-reviewed articles. Inclusion criteria were: (i) articles written in English or Portuguese (ii) articles involving adults (≥18-year-old) and (iii) full-text availability. Exclusion criteria included: (i) articles in languages other than English or Portuguese, (ii) paediatric populations and (iii) articles deemed irrelevant to the objectives of the review. (2) Discussion of a clinical case, including a patient interview, complemented by analysis of clinical records and the patient's psychotherapist input. This discussion was grounded in the findings of the literature review and informed by the patient's assessment of alexithymia using the Toronto Alexithymia Scale 20.

Conclusions

Patients with alopecia areata often present with a mixture of physical symptoms and psychological distress, requiring multidisciplinary care. Repeated ineffective treatments can delay recovery and increase emotional and financial health institutional burden. Dedicated psychosomatic and psychodermatology units are essential to integrate medical and mental health interventions, as shown in the literature and embodied in clinical case.

Resumo

Introdução e Objetivos

O objetivo desta dissertação, ilustrada com um caso clínico, é fornecer uma revisão narrativa da literatura sobre a medicina psicossomática contemporânea, com especial enfoque no domínio da psicodermatologia e numa compreensão abrangente da alopécia areata. Outros objetivos incluem: informar os profissionais de saúde – sobretudo os dermatologistas, psiquiatras e psicólogos – sobre a importância do funcionamento psicológico dos doentes e, muitas vezes da psicopatologia, nas doenças psicossomáticos e da necessidade de cuidados interdisciplinares; sensibilizar as instituições de ensino da área da saúde para a importância da inclusão de conteúdos básicos de psicossomática na formação médica, dada a sua relevância em múltiplas apresentações clínicas; promover, junto de decisores políticos e administradores de cuidados de saúde, o reconhecimento do impacto da abordagem integrada nestas patologias complexas, incluindo a melhoria da qualidade de vida, o aumento da produtividade e a redução dos custos dos cuidados de saúde a longo prazo.

Metodologia

(1) Realização de pesquisa bibliográfica de artigos publicados entre 1 de janeiro de 2015 e 1 de junho de 2025 utilizando as bases de dados PubMed Central, SCOPUS, PsycINFO e PsycNET. Os seguintes termos Medical Subject Headings (MeSH) foram usados como palavras-chave e combinados em pares: "*Alopecia Areata", "Alexithymia", "Multidisciplinary Care Team", "Psychodermatology", "Psychological Factors", "Psychopathology", "Psychosomatic Medicine"* e *"Therapeutics".* Foram também incluídos outros artigos relevantes, incluindo as referências das fontes de cada artigo usado. Todas as publicações foram pesquisadas pelo título, resumo e texto integral e a sua seleção foi feita de acordo com os objetivos supracitados. Foi dada prioridade a artigos originais, revisões sistemáticas, meta-análises e artigos revistos por pares. Os critérios de inclusão foram: (i) artigos escritos em inglês ou português (ii) artigos envolvendo adultos (≥18 anos) e (iii) disponibilidade de texto completo. Os critérios de exclusão incluíram: (i) artigos em outras línguas que não o inglês ou o português, (ii) populações pediátricas e (iii) artigos considerados irrelevantes para os objetivos da revisão. (2) Discussão de um caso clínico, incluindo uma entrevista com o doente, complementada pela análise dos registos clínicos e pelo contributo da sua psicoterapeuta. Esta discussão foi sustentada pelos dados obtidos na revisão da literatura e informada pela avaliação da alexitimia do doente, através da aplicação da escala *Toronto Alexithymia Scale* 20.

Conclusões

Os doentes com alopécia areata apresentam com frequência sofrimento físico e psicológico interligado, o que exige a sua abordagem multidisciplinar. A repetição de tratamentos ineficazes pode atrasar a recuperação e agravar o impacto emocional e financeiro. A criação de unidades dedicadas de psicossomática e psicodermatologia é essencial para integrar cuidados médicos e de saúde mental, como demonstrado no caso clínico apresentado.

Keywords

alopecia areata, alexithymia, psychodermatology, psychological factors, psychopathology, psychotherapy.

List of Abbreviations

AA – Alopecia Areata

ACE(s) – Adverse Childhood Experience(s)

ACTH – Adrenocorticotropic Hormone

AD – Autoimmune Disorder

AT / AU – Alopecia Totalis / Alopecia Universalis

BDNF – Brain-derived Nerve Factor

CA(s) – Catecholamine(s)

CRH – Corticotropin-releasing Hormone

DAA – Diffuse Alopecia Areata

DDF – Difficulty in Describing Feelings

DD(s) – Depressive Disorder(s)

DIF – Difficulty in Identifying Feelings

EOT – Externally Oriented Thinking

HPA – Hypothalamic-Pituitary-Adrenal

IL(s) – Interleukin(s)

IL-1β – Interleukin 1-β

LC-NE – Locus Coeruleus-Norepinephrine

NGF – Nerve Growth Factor

PTSD – Post-Traumatic Syndrome Disorder

QoL – Quality of Life

SLE(s) – Stressful life event(s)

SSRI(s) – Selective Serotonin Recaptation Inhibitor(s)

T_1 / T_2 / T_{17} – Type 1 / Type 2 / Type 17 (immune activity)

TAS-20 – Toronto Alexithymia Scale 20

TNF-α – Tumour Necrosis Factor-α

Vit D – Vitamin D

Objectives

The objective of this dissertation, illustrated with a clinical case, is to provide a narrative review on contemporary psychosomatic medicine, with a particular focus on the field of psychodermatology and a comprehensive understanding of alopecia areata. Additional aims include: informing healthcare professionals – namely dermatologists, psychiatrists, and psychologists – of the critical role played by patients' psychological functioning and, often times, underlying psychopathology in psychosomatic disorders, as well as the importance of interdisciplinary care; raising awareness among health education institutions of the need to incorporate concepts of psychosomatic medicine into medical training, given their relevance across a wide range of clinical presentations; and promoting, among policy-makers and healthcare administrators, the recognition of the impact of integrated approaches in the management of complex conditions, including improvements in quality of life, increased productivity, and reduced long-term healthcare costs.

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A clinical case was also collected by means of a patient interview, review of clinical records and discussion with the patient's psychotherapist. Prior to the clinical interview, several preparatory steps were undertaken: (i) the clinical and academic purpose of the work was clearly outlined and submitted, with substantiating arguments, to the hospital's Health Ethics Committee, which granted approval; (ii) the patient was initially invited to participate and was verbally informed with clear, accessible and non-technical language of the exclusively academic nature of the study and (iii) after a period of reflection, the patient provided explicit, written informed consent in full compliance with ethical standards and data protection regulations. The clinical case analyses underwent: (i) data pseudonymisation through pseudonym generation, character masking, generalisation and data perturbation and (ii) grounding in the literature review and was further informed by the patient's alexithymia assessment using the 20-Item Toronto Alexithymia Scale (TAS-20).

Introduction

Alopecia Areata

Alopecia areata (AA) is an autoimmune disorder (AD) causing episodic or chronic non-scarring hair loss, normally in a relapsing-remitting pattern. The most common form is patchy AA, with one or more round or oval patches of partial or total hair loss. This can occur in any hair-bearing area, typically the scalp but also the beard, eyebrows, eyelashes and pubic hair. Other variants include alopecia totalis (AT; total scalp hair loss), alopecia universalis (AU; total body hair loss), ophiasis (band-like loss on temporal and occipital scalp), and sisaipho (the reverse pattern). Diffuse AA (DAA, acute widespread shedding) and concentric regrowth patterns have been described[1,2]. Regarding sex, males more often report childhood onset and family history, while females tend to have a prolonged course and personal history of AD or psychiatric disorders (PDs)[3]. Hair loss is usually symptomless, however, occasionally, pruritus and trichodynia may appear in affected areas[4]. Interestingly, pruritus may precede hair loss[5] and the scalp is a common site for psychogenic pruritus, especially in persons with PD[6]. Also, trichodynia often co-occurs with emotional dysregulation, anxiety and depression. This implies that emotional stress and PDs may precipitate or exacerbate AA, possibly via cutaneous nerve hyperreactivity and mediator release at the hair follicle[4].

Psychodermatologic Classification

AA has been classified as a psychosomatic disorder[7]. Nowadays, AA is more recognised to be an AD with a psychiatric role to its pathophysiology. This stands from the fact that psychological and psychopathological factors modulate autoimmune dysregulation not as direct causes but rather via stress-mediated pathways[8]. Although no universally accepted classification system exists for psychodermatological disorders, AA may be considered to fall into 2 categories: (1) psychophysiological disorders, where psychological stress precipitates or exacerbates the dermatosis with a clear temporal association; and (2) dermatological disorders with psychiatric comorbidity, in which emotional distress arises as a consequence of the dermatosis, often excessively given the resulting physical impact[9].

Psychosomatic Perspective

Psychoneuroimmunoendocrinology

Psychological stress initiates a complex cascade of neuroendocrine and immunological responses with direct impact on multiple organ systems. The central nervous system (CNS) perceives stressors and activates both the locus coeruleus-norepinephrine (LC-NE) system and the hypothalamic-pituitary-adrenal (HPA) axis, initiating the classic "fight-or-flight" response. LC-NE system stimulates the rapid release of catecholamines (CAs) – primarily norepinephrine and epinephrine – from the adrenal medulla, which increase heart rate, blood pressure, respiratory rate and muscular tension. Simultaneously, the HPA axis prompts the release of corticotropin-releasing hormone (CRH) from the hypothalamus, which stimulates the anterior pituitary to secrete adrenocorticotropic hormone (ACTH). ACTH acts on the adrenal cortex, promoting the synthesis and release of cortisol.

The nervous and immune systems communicate bidirectionally. Immune cells, including lymphocytes and macrophages, express receptors for cortisol and catecholamines, allowing them to respond to neuroendocrine signals. Conversely, cytokines produced by immune cells – especially interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α) – can cross the blood-brain barrier or signal through the vagal nerve, modulating brain function and contributing to symptoms such as fatigue, sleep disturbances, mood alterations, and even cognitive impairment.

Under conditions of chronic stress, however, there is often a shift from a balanced immune response to a dysregulated profile. This is characterized by initial pro-inflammatory activation followed by functional immunosuppression, which might contribute to autoimmune reactivation or even the progression of chronic inflammatory diseases.

Lastly, emerging research suggests that the brain may encode and retrieve "immunological memories" of past inflammatory responses. These can be reactivated by psychological triggers, potentially leading to recurrence of somatic symptoms in the absence of actual pathology – highlighting a mechanism for psychosomatic perpetuation of conditions such as psychodermatologic disorders[10].

Psychoanalysis

Since the beginning of 20th century psychoanalytical studies have given great interest in somatic symptoms and its relationship with psychoemotional issues.

Skin is an organ highly reactive to mental affective states. From a psychodynamic perspective, the skin may express negative emotions or unresolved emotional conflicts that are difficult for the patient to perceive, interpret or express. Thus, only by also addressing psychogenic factors can effective treatment be achieved in dermatoses with significant psychosomatic components[11].

Hair and Hair Loss Symbolism

Historically, human hair has been used to depict one's beauty and strength. By contrast, its loss would represent some sort of punishment of a weak, if not evil, moral character. Alopecia has been connected the eroding of the inner self trough the physical substance and the sufferer stands out to everyone else, involved in shame and incompleteness[12]. Many meanings are linked to hair: biologically, it reflects health and psychic well-being; socially, it signifies class status, ideals of beauty, cultural belonging and even mystical power; psychologically, it serves as an object of human relationships, a site for resolution (e.g. cutting, growing) and a marker of self-transformation[13]. Hair also carries unconscious associations with parental figures: as the locus of maternal desire, it reflects the need for care and the desire to attract affection with its loss reflecting detachment from primitive affection or an inability to fulfil nurturing impulses; as a paternal symbol, hair embodies strength and maturity, with its loss evoking fears of emasculation or conflict with the paternal role[14].

The Phenomenon of Somatization

An integrative view suggests that negative life events accumulate as latent affective energy, remaining embedded throughout an individual's life course. In the context of AA patients – discussed in detail below – such energy nuclei often stem from early traumatic experiences, including loss of relatives, parental rejection and insufficient attention during childhood. Subsequent exposures to analogous stressors or emotionally similar experiences may reactivate these energy clusters. Then, the unconscious mind tries to symbolize the reactivated experiences by attributing meaning to them. Failure to do so may result in the manifestation of neuropsychiatric symptoms at the neuropsychic level. Conversely, successful symbolic integration may trigger a biological response via neuroimmunoendocrine pathways, potentially leading to the development of somatic disease and the formation of immunological memory[15].

In a related framework, Schur postulates that the Ego operates through two modes of thought organization, conceptualized as bound energy available for autonomous regulation: primary process thinking – a more primitive, pre-Ego response – and secondary process thinking – representing a mature, regulated cognitive response. Schur argues that bodily homeostasis requires the neutralization of primary process energy, transforming it into secondary process thinking and thus facilitating the desomatization of stimuli responses. Somatization – or more precisely, resomatization – occurs when there is a physiological regression from secondary back to primary process thinking, leading to the emergence of somatic symptoms[16].

In alopecia areata, hair may serve as a symbolic locus for processing traumatic life events. The individual's symbolic interpretation of trauma—such as a perceived loss of identity—may align with culturally and personally ingrained symbolic meanings of hair (e.g., identity and selfhood), thereby directing the neuroimmunoendocrine response specifically to the hair follicle[15, 16].

Ego as a Primitive Defensive Mechanism

Anzieu's Skin-Ego theory proposes that psychic contents are derived, as a starting point, from body surface experiences and that this transposition process remains throughout the rest of the human life. The author proposes a multilayered model of the Self: an outward porous layer, resembling a mesh, individualizes the Self's style and character while filtering exchanges with the world; an inner resistant layer, akin to muscle, protects Self's identity. Between them lies the Ego, acting as a flexible interface and psychic defence, which regulates the access of the outside world to the contents of the Self[17].

Family Dynamics in Psychosomatics

Communication between two human beings can only be achieved through the perception, or the idea, of an object given that the object itself is out of the reach of the human mind. This interchange of the object can be, for instance, symbolized by a symptom of a disease that the external observer can perceive. AA often seems to convey an ambivalent meaning of the communicated idea. On the personal level, the person means to cause attraction, wanting to be seen by others, yet at the same time intends to cause repulsion, not wanting to be touched by others. This underlies a desire of close interpersonal relationships that is refrained by an uncertainty about intimacy and commitment which can be internalized, for instance, if the person grows up associating the idea of hair, notably if long and well-treated, as symbolizing the object of attractiveness. Concerning the family, its autonomy seems to be organized through the idea of closeness, when showing concern about the illness, yet also by distance, when limiting other interactions with the sufferer. This idea can arise if the individual grows up to associate behaviours of detachment, such as rows, assaults and separations, as a meaning of what a relationship is supposed to be[18]. By representing a somatic symptom, the hair can have a double role of reinforcing and of contradicting the internal conflict. This observation is in accordance with Schur's perspective, when the author finds a common characteristic of patients with psychodermatological disorders to be the extreme ambivalence in their objects of symbolization[16].

Associated Factors in AA

Predisposing Factors

Adverse Childhood Experiences

Adverse childhood experiences (ACEs) may lead to chronic inflammation during adulthood.

On one hand, early-life mental trauma correlates to a state of low-grade systemic inflammation, which may contribute to the pathogenesis of ADs. In fact, a meta-analysis found a significant association between ADs – especially localized diseases such as AA – and three specific forms of ACEs: emotional abuse, emotional neglect and domestic violence exposure[19].

On another hand, childhood mental trauma has also been linked to the emergence of clinical or subclinical psychopathology. Specifically, another meta-analysis proposed that alexithymia, when established during early developmental stages, may partially mediate this relationship. Experiences of emotional abuse, emotional neglect and physical neglect appear to exert a great influence on primary alexithymia development. This personality construct is thought to emerge, at least in part, as a maladaptive coping strategy due to the absence of emotionally responsive caregiving – particularly from primary attachment figures such as parents, who typically model and reinforce emotional communication during childhood[20].

Taken together, these findings suggest that childhood trauma, particularly childhood neglect, may contribute to the later development of AA and comorbid PDs.

Stressful Life Events

Patients frequently report SLEs prior to the onset or, more often, prior to an exacerbation of AA, either in early childhood or later stages of life[21,22,23].

During childhood, mainly around 8 to 9 years of age, emotional neglect (e.g. being left alone, scarce affection) and emotional abuse (e.g. being belittled, threatened verbally or unjustly punished) within the family unit as well as the loss of a family member seem to be the primary scenarios[21].

During adulthood, physical damage (e.g. injury, illness) and changes in sleep (e.g. working in shifts), personal (e.g. dressing, manners) or eating habits, are more strongly correlated[23]. Interestingly, in societies with patriarchal structures, women may report more personal problems whereas men may correlate more to financial ones[24].

Biologic and Cognitive Factors

Psychoneuroimmunoendocrinology

As previously stated, the major neuroendocrine stress pathways, LC-NE system and HPA axis, may represent the common aetiology linking behaviour and some inflammatory immunodermatoses[25]. Importantly, it is believed that these stress systems may modulate higher reactivity to stressful life events (SLEs) primarily in people with particular vulnerabilities (e.g. anxiety-prone traits, limited social support)[22,24]. As an example, it has been theorized that COVID-19 stressors – such as fear, isolation or post-traumatic symptoms – may have triggered more frequent AA outbreaks and relapses in patients with high pre-existing stress (e.g. medical students, people with previous mild mental illness)[26, 27].

Regarding acute emotional stress, it is readily known that LC-NE system activation increases CAs availability – particularly norepinephrine – leading to a heightened cholinergic tone. In AA patients, evidence suggests that, due to the reduction of blood flow and impairment of endothelial or autonomic functions, the result may be a fatigue-related microcirculatory dysregulation in hair follicles, a process that may be triggering for the disease regardless of its activity or severity[28]. The continuation of such acute stress exposure in these patients will attenuate, in a compensatory manner, HPA axis activation. Indeed, major naturalistic stress exposure over a period of 4 to 12 weeks has been associated with increased type 1 (T_1) immune activity in hair follicles – marked by a peak in tumour necrosis factor-α (TNF-α) during the inter-stress period – and a blunted diurnal rise in salivary cortisol[29].

By contrast, chronic emotional stress activates the HPA axis and triggers CAs release, leading to higher intestinal permeability and lower neural dopamine and serotonin levels[30]. Additionally, brain levels of neurotrophins such as nerve growth factor (NGF) and brain-derived nerve factor (BDNF), both linked to psychiatric disorders, also diminuish[31]. This overall phenomenon can be further potentiated by vitamin D (Vit D) deficiency[30], since low serum active Vit D levels mediate low serum BDNF levels that, on their turn, are associated with depressive symptoms[32]. Specifically, Vit D deficiency impairs its neuroprotective role in calcium regulation and neuronal gene expression as well as melanin synthesis and hair growth in the skin, and systemically disrupts immunomodulation, serotonin and BDNF production[32].

In the longer term, however, chronic stress exposure – which induces higher CRH receptors expression in mononuclear cells[25] and higher levels of CAs and metabolites[33] (e.g. metanephrine) – is actually linked to lower activity of adrenocorticotropin receptors[8]. This reflects an exhausted HPA axis, marked by a high psychological stress perception and, paradoxically, by a lower physiological stress response. Similarly, AA patients with high subjective anxiety may have reduced expression of the translocator protein – associated with the physiological stress response – showing that a vulnerable anxiety-prone personality trait, much more than transient periods of acute anxiety, is also an important factor modulating the onset and severity of AA[34].

Immunological Profile

In AA, tissue and circulating levels of inflammatory interleukins (ILs) are elevated leading to the activation of T_1, type 2 (T_2) and type 17 (T_{17}) immune pathways[35]. This distinct immune profile may help explain the frequent co-occurrence of AA with psychopathology. To begin with, activation of T_1 pathway increases, among other, the production of interleukin 1-β (IL-1β) and tumour necrosis factor-α (TNF-α), promoting oxidative stress in hair follicles and differentiation of T_{17} cells[35]. In parallel, these molecules seem to decrease neural dopamine availability[30] and elevate glutamatergic neurotoxic products[35], respectively. Additionally, severe forms of AA display a molecular signature that induces activation of T_2 and T_{17} pathways, responsible for an increase in IL-22 and IL-25 serum levels, respectively. These cytokines may contribute to the higher incidence of depression observed in AA patients, possibly by binding to receptors on neural and brain endothelial cells[35].

Interestingly, juvenile-onset AA may be linked with a more severe disease course compared to adult-onset forms, possibly because chronic psychological stress promotes the development of T_{17} immune cells. Specifically, this population secretes IL-17 cytokines that induce the production of pro-inflammatory mediators collectively contributing to the organ-specific chronic inflammation of AA. Particularly, it has been proved that serum levels of IL-17A – positively correlated with poorer prognosis – were significantly elevated in younger patients (under 30 years of age) with recent-onset AA (disease duration less than 6 months). One possible explanation is that younger persons, due to their ongoing psychological development, may have fewer coping strategies to manage chronic emotional stress, rendering them more vulnerable to stress-related immune dysregulation[7].

Lastly, disease activity appears to be linked to increased comorbid psychiatric disorders (PDs), possibly mediated by elevated IL-33 produced by T_2 immune cells. In a study, while all AA patients exhibited higher rates of psychopathology, overt clinical psychosis was stated only in a few with active disease. Besides, active cases also had higher IL-33 levels, which correlated positively with disease chronicity and severity[8].

Psychophysiology

AA patients show increased hypothalamic activity, suggesting stress-related dysregulation from chronic psychological tension. The hypothalamus also displays stronger connectivity with the left postcentral gyrus – implicated in emotional and somatic processing – and with the right inferior temporal gyrus – involved in emotion-cognition integration. It is possible that greater hypothalamus-postcentral gyrus coupling is linked to more severe depression, reflecting maladaptive stress responses, whereas altered connectivity with the inferior temporal gyrus correlates with lower immune activation, possibly indicating compensatory mechanisms[36].

Psychological, Social and Cultural Factors

Psychological Profile

These patients seem to have a distinct psychological profile marked by higher harm avoidance – a tendency toward cautiousness and pessimism, social withdrawal and greater fatiguability – and higher reward dependence – pointing to high emotional sensitivity and greater need for social attachment and approval[37]. Indeed, the greater experience of emotional reactivity and negativity leads to a greater conflict within their social context[38].

Metacognition

Given the association of AA with psychiatric diseases, maladaptive metacognitive control strategies may contribute to the occurrence and continuation of this disease, especially if not mitigated by adequate coping skills. Indeed, a study found that AA patients have some problematic adjustments when it comes to cognitive events and processes of their minds. For once, the need to control intrusive thoughts showed to be significantly higher, which may be linked to reduced self-perception and cognitive self-confidence when assessing their own toughs. Also, the cognitive self-consciousness, in other words, the beliefs in rigid and negative toughs about the disease, was also found to be significantly higher in persistent cases, which could amplify stress levels even further and cause the maintenance, or at least the recurrence, of the disease. Similarly, higher number of thoughts related to danger and uncontrollability of the disease, known predictors of obsessive-compulsive symptoms and pathologic anxiety, were correlated to a greater degree of disease involvement[39].

Emotional Processing

Distress tolerance can be diminished in AA patients[40] and may be partially attributed to an impaired emotional intelligence in coping with daily stressors and relationships[41]. Such deficits may contribute not only to stress-related symptoms (e.g. tension, indecisiveness and somatic complaints) but also greater psychological suffering, a mild depressive reaction to the disease and a heightened risk of psychiatric comorbidities[31,40,41]. In AA patients, with emphasis on cases with comorbid pruritus, a difficulty in the expression of anger, both towards oneself and towards the others, seems to predominate and, biologically, this may contribute to an increase in sympathetic tonus and somatization[42]. This repressed anger manifests trough higher anger-related symptoms (e.g. triggered by being criticized, ‘‘not being taken seriously’’), anger-associated thoughts (e.g. facially trough a hostile outlook, physically trough outward anger) and interpersonal anger-related behaviour (e.g. passive-aggressive reactions)[43].

Illness Perception

In chronic diseases, the cognitive and emotional interpretations of the experienced illness can, influence coping strategies, health outcomes and quality of life (QoL). In AA, this impact seems to be higher in patients with stronger beliefs about the disease's impact, higher illness identity and negative emotions. This is also true for patients who attribute the disease to internal or external causes, rather than to bad luck or chance, possibly due to an attributed self-responsibility or a perceived need for lifestyle changes. Finally, avoidant coping (e.g. denial, self-distraction, substance use) – with thought suppression paradoxically increasing thought frequency – and problem-focused coping (e.g. planning, religion) – where control over disease is minimal – may offer limited benefit in unpredictable conditions like AA[44].

Stigmatization

Patients with AA often face disease-related stigmatization due to the visible nature of hair loss, which deviates from social norms and damages their perceived identity[45,46].

Social stigma derives from general unawareness of AA leading to prejudiced attributions (e.g. unattractiveness, contagiousness) and behaviours (e.g. bad judgment, discriminatory treatment)[46,47]. This marginalization phenomenon is further stereotyped by media coverages – often depicting AA as an unprioritized condition of a social minority[45] – and some behaviours of health professionals – such as doctors, nurses, and technicians[48].

Self-stigma is linked to lesion visibility (e.g. socially or sexually involved regions) and negative self-perceptions (e.g. embarrassment, rumination)[47,49]. This often implies a cognitive effort to suppress automatic disease-related reactions, which might signify that AA people suffer more from an internalized "hidden" self-stigma rather than to a truly perceived social stigma[50]. High self-stigma (e.g. self-worth, sensitivity to judgment) is strongly associated with poor QoL[51] and social withdrawal, ultimately worsening overall mental health[46,47] and increasing depressive symptoms and suicidality[48].

Alexithymia

Alexithymia, meaning “no words for emotion”, is a psychological construct which was firstly intellectualized by Sifneos to describe emotional difficulties in psychosomatic patients[52]. It comprises three core components: (1) difficulty in identifying feelings (DIF), which impairs the discrimination of true feelings from physical signals aroused by them, (2) difficulty describing feelings (DDF) to others and (3) a limited imaginative capacity supplanted by an externally oriented thinking (EOT) style[53,54]. Taylor emphasises that this emotional processing deficit leads to poor symbolic representation of affective states, meaning that patients cannot mentally represent or communicate their feelings. Because of this, emotions become "somatised", manifesting as physical symptoms rather than being experienced or verbalised psychologically, which disrupts psychobiological regulation and contributes to psychosomatic disorders[55]. Alexithymics also show hypervigilance and hyperarousal to physical stimuli, due to heightened somato-visceral and neuroendocrine responses, and tend to feel negative emotions, partly driven by harsh self-criticism and poor social relations[53,56].

The “stress-alexithymia hypothesis” suggests that deficient awareness of emotions, inability to express and verbalize affects plus difficulty differencing psychological from physical sensations – the cognitive component – lead to ineffective/maladaptive coping efforts – the behavioural component – fostering the already increased stress exposure and the inherent autonomic hypersensitivity – the physiological component. Some authors even consider it a disorder of cognitive-affective processing since alexithymics don’t seem to recognise somatic sensations as being physiological indications of experienced feelings and misinterpret them as being illness symptoms. Consequently, people with pronounced alexithymia are more vulnerable to somatic and affective disorders and may respond differently to treatment[53-56].

In AA, alexithymia may be more prevalent among women and patients with pruritus[57]. It may represent a stable vulnerability trait – alexithymia-trait – particularly in patients exposed to early-life stressors, impairing emotional development and expression long-term. Alternatively, it may represent a reactive state – alexithymia-state – emerging in response to AA-related distress, given its strong correlation with dissociative symptoms (e.g. body detachment). Thus, it may reflect a psychological escape when emotional processing becomes overwhelming, making it a defence mechanism against the burden of hair loss, especially in severe cases[58].

Psychiatric Comorbidities

There is a strong link between psychiatric, psychological and dermatological conditions: mental disorders may be the result of a severe and disfiguring skin disease – a primarily dermatologic condition – or mental disorders can be the direct cause of a skin disease, manifesting initially with skin-related symptoms – a primarily psychiatric condition[56]. In the first case, patients are likely to develop new-onset PDs (e.g. adjustment disorder, panic disorder, sexual dysfunction) within the first year of diagnosis[59,60] and typically affective symptoms improve over one year of treatment[61]. Conversely, patients may have pre-existing PDs (e.g. anxiety, sleep disturbances, depression)[59,60], with psychiatric care prior to the disease onset being indicative of this[61].

AA patients have higher lifetime prevalence and incidence of PDs, especially women, adults aged 30-49 and white individuals[60,62]. This association is stronger for anxiety and depression and to a lesser extent for adjustment disorders and post-traumatic syndrome disorder (PTSD). However, regarding anxiety and depression, subclinical symptoms, which may progress into clinical disorders, are more common that full-blown diagnoses meeting diagnostic criteria. This emphasizes the need for early detection with screening tools, preferably clinician-administered assessments, which tend to identify more symptoms – possibly due to the inclusion of somatic items in depression scales[63]. Finally, an increased risk for somatoform disorders exists if AA is associated with pruritus, possibly due to intensified sensory focus and scratching behavior[64].

It is also worth noting that, in AA, psychiatric comorbidities link to more severe phenotypes – beyond scalp hair loss, poorer disease course and need for more aggressive treatments[65]. Additionally, disease relapses correlate with more severe psychiatric manifestations, ranging from neurotic traits (e.g. anxiety, obsessive-compulsive tendencies) to psychotic-like features (e.g. paranoid ideation, psychoticism), challenging the idea that emotional adaptation develops with repeated episodes[37].

Depressive Disorders

Depressive disorders (DDs) in AA patients tend to manifest with anhedonia, fatigue, reduced appetite and impaired concentration[66], with higher risk groups including those with onset under 20 of age, involvement of eyelashes and/or eyebrows, poor therapeutic response, embarrassment and social deprivation[67,68].

Both conditions may share a synergistic pathomechanism, stemming from common risk factors, immunological pathways and environmental factors[66,67]. Firstly, familial aggregation of AA and MDD has been found, with unaffected siblings of affected individuals with either condition having a higher risk for the other. This is more significant when AA precedes MDD and among female probands and female siblings, possibly reflecting women's greater tendency to negative cognitions and emotional responses to loss. Likewise, polymorphisms in the macrophage migration inhibitory factor gene have been associated with early-onset extensive AA and antidepressant-responsive MDD. This genetic overlap may involve the MHC region and represent a distinct MDD phenotype, or cluster of specific symptoms, in AA patients[69]. Secondly, systemic inflammation may potentiate the development of DDs at the beginning and during the progression of ADs[66]. In reverse, affect can be implicated in cell-mediated immunity, so DDs may also contribute to ADs[67]. In fact, the gene GRM8 – which encodes proteins of glutamatergic synapses of neuronal communication and excitatory signalling – has significant genetic variation in both diseases, so it can be hypothesized that dysregulation of glutamate synapses might contribute to their co-occurrence[70]. Finally, this link may also involve impaired conversion of homocysteine to cysteine, dependent on vitamins B6, B9 and B12. This leads to elevated homocysteine – predisposing to oxidative stress, neurotoxicity and depression – and reduced cysteine/glutathione levels – waning antioxidant defences in hair and brain[71].

Both diseases also follow a bidirectional pattern over time. The mean time for AA to precede depression is 4 years shorter than the mean time for depression to precede AA, suggesting that AA may

be a proximal risk factor for the development of depression, whereas depression represents a more distal risk factor for future AA[72].

Anxiety Disorders

Although acute stress episodes have a stronger link with AA[73], anxiety is also very prevalent – ranging from subclinical symptoms to undefined and generalized anxiety disorders[74]. This may result from fears related to the disease or its recurrence, as well as persistent concerns about health, living conditions and social acceptance[56]. A systematic review with meta-analysis found a high prevalence of social anxiety disorder and separation anxiety disorder, with a less evident association concerning specific phobia and obsessive-compulsive disorder[63]. AA has also been linked to PTSD, particularly in younger people, and to higher odds of alcohol use – from alcohol abuse to alcohol use disorder – resulting from comorbid psychological and psychiatric comorbidities.[75]

Body Dysmorphic Disorder

More than 10% of patients with dermatological diseases experience symptoms suggestive of body dysmorphic disorder (BDD). Regarding AA, patients show an at least 11-fold increased risk of clinically significant BDD symptoms. Given the societal ideal of flawless skin, social stigmatization – especially through confused or hostile reactions from others – emerges as the strongest predictor of this pathology, surpassing depression, anxiety, and suicidal ideation. Such dynamics may inclusively explain why patients often conceal BDD-related symptoms during dermatology consultations[76].

Sleep Disorders

A systemic review found a bi-directional cause-effect relation between AA and sleep disorders, mostly in individuals under 45 years of age[77]. On one hand, AA patients have higher risk for developing new-onset sleep disorders, both obstructive sleep apnea and nonorganic insomnia. Conversely, sleep quality and sleep disorders correlate with the severity of AA. Indeed, circadian clock disturbances, for instance deficiency of *Clock* or *Bmal1* genes, are known to raise the risk of autoimmunity as well as delay the anagen progression of hair cycle[78]. Other studies found that in AA patients impaired sleep quality (e.g. shorter sleep time, excessive daytime sleepiness) is an independent factor linked with higher mood disturbances (e.g. anxiety, depression), worse QoL and type D personality prevalence[77,79].

Psychosexual Disorders

Sexuality is an integral part of human physiology and emotional intimacy. First and foremost, self-perceived unattractiveness, typical of chronic dermatological diseases, can trigger shame and fear of rejection ultimately leading to intimacy avoidance and, potentially, partnerships breakups. Then, DD(s) may impair various sexual functional domains – desire (e.g. reduced libido), arousal (e.g. erectile dysfunction, vaginal dryness) and satisfaction – often worsened by antidepressant side effects. Lastly, anxiety disorders can amplify fear-related cognitions (e.g. negative evaluation, social rejection), often leading to social withdrawal and dissolution of interpersonal relationships. These issues may be intensified in homosexual and bisexual men, both on a personal and on a community level, given the high aesthetic standards often met within these subcultures[80].

Obsessive-compulsive Disorders

It also has been found a significant link between cutaneous ADs, including AA, and obsessive-compulsive disorders, with an incidence 6 times higher in this population for both sexes and all age groups. This may result from a combination of a common genetic factor within families and a dysfunctional immune system, marked by increased inflammatory cytokines and imbalance and dysfunction of regulatory T cells[81].

Personality Disorders

Type C personality disorders may also co-occur with AA. Indeed, since people with avoidant, dependent and obsessive pathological traits depict a strong need of approval, care and self-control, respectively, this personality cluster often ties up with a greater interiorization of stress and worst adjustment to illness, worsening the illness prognosis[56]. Eventually, AA relapses can even pose a risk to the emergence of paranoia and obsessive-compulsive traits[68].

Suicidality

Patients with AA show an increased incidence of suicidality. At one end of the spectrum, young adult men with rapid-onset hair loss represent a particularly at-risk group for self-harm[68]. At the other, individuals with severe forms of AA[56] – chiefly those with comorbid alexithymia[38] – report higher rates of suicidal toughs and attempts. AA itself poses an independent risk factor for the suicidal behaviour continuum, regardless of sex, age or psychopathology. This is possibly derived from the heightened neuroendocrine reactivity, marked by central HPA axis hyperactivity and peripheral corticosteroid resistance, commonly observed[82].

Psychologically Amplified Neurological Conditions

AA may increase the risk of both dementia and stroke. Significant associations have been found between AA and Alzheimer's disease as well as unspecified dementia, with affected individuals diagnosed on average 5 years earlier than controls. This link appears bi-directional, since AA-derived psychosocial stress may reduce both social and physical activity – known dementia risk factors – while shared inflammatory pathways – like increased IL-6 and TNF-α – may contribute to neurodegeneration and cognitive decline[83]. Similarly, AA imprints a higher risk of stroke, not only due to comorbid traditional cardiovascular risks – hyperlipidaemia, coronary heart disease and obesity – but also because chronic inflammation and depression may further promote cerebrovascular disease through atherosclerotic mechanisms[84].

Therapeutic Approaches

Given the overlap between AA and pathopsychological comorbidities, well-tolerated medical and psychological treatments may benefit both. Indeed, mind-body interventions can improve psychiatric wellness, psychosocial functioning and, in some cases, hair regrowth derived from modulation of stress and inflammation[59,85]. This highlights the importance of a multidisciplinary approach, particularly when medical therapy alone proves insufficient.

Psychosomatic Evaluation

Firstly, one should examinate the personal emotional experience (e.g. perceived precipitating and perpetuating factors, model of illness, self-image) and the broader psychosocial impact (e.g. relationships, occupational/academic functioning) of the disease[86]. It is noteworthy that AA patients are often very sensitive to the clinician's attentive listening and authenticity[87]. Other aspects include assessing childhood trauma in patients with alexithymic traits[20], screening for psychiatric comorbidities using brief validated tools[75] and monitoring sleep and cognition (e.g. dementia screening from age 45)[83]. After this evaluation, psychotherapeutic and, if indicated, psychopharmacological interventions should be offered.

Dermatologic Treatments

Current dermatologic treatments for AA primarily target immunosuppression or, at least, immunomodulation of disease activity. However, existing treatments have unsatisfactory responses since they associate with high relapse rates, particularly in severe cases, appear ineffective in altering the long-term disease trajectory. The best evidence-supported treatments for AA include topical corticosteroids (e.g. betamethasone) to reduce local inflammation, intralesional corticosteroids (e.g. triamcinolone) injected directly into lesions to suppress immune activity and topical immunotherapy (e.g. diphencyprone) which induces local allergic contact dermatitis to modulate immune response[2].

Psychotherapeutic Treatments

A full course of cognitive behavioural therapy, typically 5 to 20 structured time-limited sessions, has demonstrated efficacy in managing stress-related aspects of dermatological disorders, with notable benefits especially in women[88]. The first step is to conceptualize the patient's dysfunctional cognitive model of illness, as earlier explained, the maladaptive beliefs about their disease – such as distorted perceptions on its aetiology, consequences, duration and controllability – that might fuel emotional dysregulation (e.g. anxiety if perceived as threatening, anger if construed as unfair) and defensive or avoidant behaviours. Is also crucial to identify negative automatic thoughts and behavioural patterns triggered by social or emotional contexts (e.g. repetitive scratching to relieve tension, hopelessness after perceived judgment). Moreover, AA patients commonly show rigid cognitive schemas focused on loss and disfigurement and their reconstruction involves cognitive strategies (e.g. challenging beliefs, reframing automatic thoughts) combined with progressively challenging behavioural experiments (e.g. guided imagery, putative social scenarios, real-life exposures, feedback from trusted others). Finally, psychoeducation of the disease – addressing disease's pathophysiology, realistic expectations, lifestyle adjustments and strategies to reduce uncertainty – should always be offered[89].

Psychodynamic approaches techniques can also be applied. A peer-reviewed expert opinion highlights that containment and identity issues of the Ego seem to be highly prominent concepts in AA that should be addressed[11]. Therapeutic relationship and active listening are critical factors in the clinical psychodynamic approach to these patients. Given the characteristics of the hospital setting, focal and brief psychotherapies are used[90].

In parallel, patients must be supported in developing adaptive coping mechanisms – such as tracking psychological triggers, reducing harmful habits and shifting attention away from symptoms[89]. A peer-reviewed study stresses that active, cognitive and social coping strategies are the most effective

ones in AA. Active coping includes cosmetic aids (e.g. wigs, makeup), stress management (e.g. exercise, meditation, nature) and emotional expression (e.g. humour, open discussion). Cognitive coping focuses on reframing identity and seeking reliable information[91]. Social coping is promoted trough peer support groups (e.g. sharing experiences, prosthesis guidance)[86] and open communication with close others[91].

Psychopharmacologic Treatments

Psychotropics should be considered when stress or neuropsychiatric disorders are significant contributing factors to AA and, eventually, when psychosocial distress becomes overwhelming. In such situations the dermatologist may play a key role in initiating treatment, as patients often decline referral to mental health services[92]. Reported agents associated with significant hair regrowth in AA cases with comorbid anxiety disorder, depressive disorder or dysthymia include selective serotonin reuptake inhibitors (SSRIs) – paroxetine (20 mg 1d) and citalopram (20 mg 1d) – and tricyclic antidepressants – imipramine (75 mg 1d) and trimipramine (100 mg 1d) [92,93]. Their therapeutic effects – alleviating psychiatric symptoms and reducing skin lesions – typically start within 3 to 6 months and seem to be more prominent with higher dosages[93].

Notably, psychiatric drugs may also induce hair loss – mainly in middle-aged women and patients with prior AA or pluri-medicated PDs – 1 to 2 months after initiation[94,95]. This incidence is highest with anticonvulsants (e.g. lithium, valproic acid), intermediate with antidepressants (e.g. bupropion, venlafaxine) and lowest with antipsychotics (e.g. haloperidol)[95]. Among SSRIs, fluvoxamine has the highest risk, followed by fluoxetine – also linked to trichotillomania – and sertraline, while citalopram, escitalopram and paroxetine carry the lowest risk[94].

Illustrative Clinical Case

Medical History

H. is a 49-year-old male who works in the automotive sector with a history of AA since age 5. Diagnosis followed patchy scalp hair loss, partially responsive to one year of oral dermatologic treatment. He had recurrent relapses and remissions over the years but did not seek regular medical care.

In his mid-30s, he presented to a dermatology centre with worsening scalp AA, coinciding with a period of high psychological stress marked by the birth of a second child and marital conflict. A psychiatric referral was suggested but declined, as he didn't perceive a link between his life events and disease flare-up.

in his mid-40s, he returned due to a substantial exacerbation of AA characterized by greater scalp hair loss (loss area ~30%), patchy beard loss and complete eyebrow tail loss. This episode occurred during a period of acute psychoemotional stress, triggered by a confrontation with his eldest daughter regarding her total school absenteeism over several months, and, days later, by finding her unconscious following a medication overdose. He recalls reacting "in despair" (*sic*) starting to scream at her, shake her body and induce her vomit. In the following dermatology consultation, H. acknowledged great psychological distress and accepted mental health referral.

At psychiatric assessment it was found depressive humour, anhedonia most of the days, feelings of worthlessness, generalized fatigue and initial insomnia, consistent with an initial diagnosis of MDD. He was started on escitalopram 5mg (1+0+0) and alprazolam 0,25 mg (0+1+2). After a week, escitalopram was increased to 10 mg (1+0+0) and, due to daytime sedation, alprazolam was reduced to 0.5 mg at bedtime (0+0+2) with a plan for discontinuation in the following 2 weeks. At follow-up, antidepressant treatment was maintained as part of the pharmacological management plan, and the patient was referred for regular psychological sessions.

Psychotherapeutic consultations consisted of monthly 1-hour sessions, combining both psychodynamic therapy and interventions focused on self-esteem enhancement. Clinical records show that H. attended all scheduled sessions consistently and that the therapeutic alliance strengthened progressively over the therapeutic journey.

At the current interview, conducted four years after initial referral, H. presents with partial scalp hair loss (loss area ~10%), partial beard regrowth and full recovery of the eyebrows. When asked, he reports an improved mood and reduced psychological distress, with occasional ruminations related to his appearance as his only complaint. H. acknowledges his personal contribution to the recovery process only when externally encouraged and admits to taking escitalopram irregularly, "only during difficult moments" (sic). His current assessment using the Toronto Alexithymia Scale-20 (TAS-20) yielded a score of 62, consistent with borderline alexithymia (see Figure 1, Appendices).

Psychosocial History

H. is the middle child of 3 siblings, having 2 sisters (see figure 2, Appendices). He describes his childhood as happy and free, marked by frequent outdoor play. While he had a strong emotional bond with the paternal grandparents, with whom he had a daily contact, the maternal ones were distant.

He started school at 5 years of age, which he now believes to have been "premature" (*sic*). Notably, he spontaneously says that "they gave me the backpack and said go", later explaining that he felt little parental support during this transition. He recalls being teased by peers, who called him "the bald one" (*sic*), but never telling anyone about it. At home, he was the sibling with most household tasks, admitting that he felt under a high level of responsibility from an early age.

He describes his father as a strict demanding figure who withheld verbal praise, which he associates with being the only male child. His mother, on the contrary, was a very passive element and would never contradict what the father decided. When directly asked, he admits having an internalized anger regarding this matter that he never expressed.

Around the age of 10, H. started to help his father in agriculture plantations throughout the year "including in school holidays" (*sic*), while his sisters would be free. Around this age, he witnessed his parent grandfather die while holding his hand, a moment he simply describes as "difficult" (*sic*).

At age 19 he enrolled in a bachelor's degree in communication sciences but discontinued after one year due to discontent with the "theoretical nature" (*sic*) of the program. He then attended a bachelor's degree in law, which he completed only after eight years because of feeling "lost and unmotivated" (*sic*) during this period. Subsequently, he began but did not finish a postgraduate course in forensic legal studies, due to "demoralization" (*sic*). He worked as a lifeguard during his studies, initially saying it was to reduce financial burdens but later realizing it was a means of gaining independence. Despite his training, he made few attempts to enter his professional field, initially attributing this to external factors but later considering personal avoidance as a factor.

He reports persistent difficulties in relationships, from primary school to university, describing himself as insecure and self-critical despite thinking to be liked by others. He had a 20-year relationship with his ex-wife, having two daughters in common, with whom he had a difficult divorce 2 years ago. He met a new partner some months ago. In daily life he struggles with decision-making, justifying it with both fear of rejection and perception of "not being given the just value" (*sic*) by others.

Comments and Reflexions

Throughout the present interview, H. repeatedly attributed his AA exacerbations to external stressors (e.g. interpersonal conflicts, family crises) and only when directly asked would he try to describe his feelings, often with a poor content (e.g. using only one word). This, respectively, reflects an external-oriented though and a difficulty in identifying and evaluating emotions which is consistent with the measured alexithymic punctuation on TAS-20 questionnaire. He also had repressed anger that showed implicitly throughout the interview but only verbalized when he was encouraged. Moreover, H. seems to have a very poor self-esteem, giving the constant need for external reassurance.

Interestingly, the clinical course of H. reflects the complex interplay that exists between immunodermatoses such as AA and psychosocial adversity, often beginning in early childhood. For once, these experiences occurred within a broad range of ACEs – notably emotional neglect, premature school entry without adequate parental support and a high burden of household responsibilities. Also, H. recalls to be exposed to social stigma early on, as evidenced by peer bullying ("the bald one"). Within the family unit, the patient's relationship with his father – a critical, emotionally withholding figure – alongside a passive maternal presence, suggests lack of affective containment during formative years. Also, witnessing the death of his close paternal grandparent at age 10, with whom he shared a strong emotional bond, may have further disrupted emotional development and coping mechanisms.

From a psychodynamic perspective, symbolic roles of hair – such as identity and autonomy – are particularly salient in this case. This and the fact that exacerbations of AA occurred during periods of heightened emotional conflict – especially those involving his parenthood – suggests a fragile ego-skin boundary, possibly akin to the concept of a pathologically porous Skin-Ego as described by Anzieu.

From a psychopharmacological perspective, the selection and titration of escitalopram (from 5 mg to 10 mg) is an excellent choice, as it carries a relatively low risk of iatrogenic alopecia and its therapeutic concentration is equivalent to that of well-established psychotropics for this purpose. Nonetheless, despite the pharmacological support, it is important to acknowledge – particularly given the low dosage and rapid improvement observed – the role of the psychotherapeutic support, to which

the patient adhered fully. This process, indeed, has enabled him to elaborate on and express emotions, and, importantly, to actively engage in the mental exploration of the meanings behind his experiences.

It is worth also noting that the clinical interview, conducted four years after the initial referral, happened during a phase of marked therapeutic recovery. This context helps explain the borderline score obtained on the TAS-20 questionnaire.

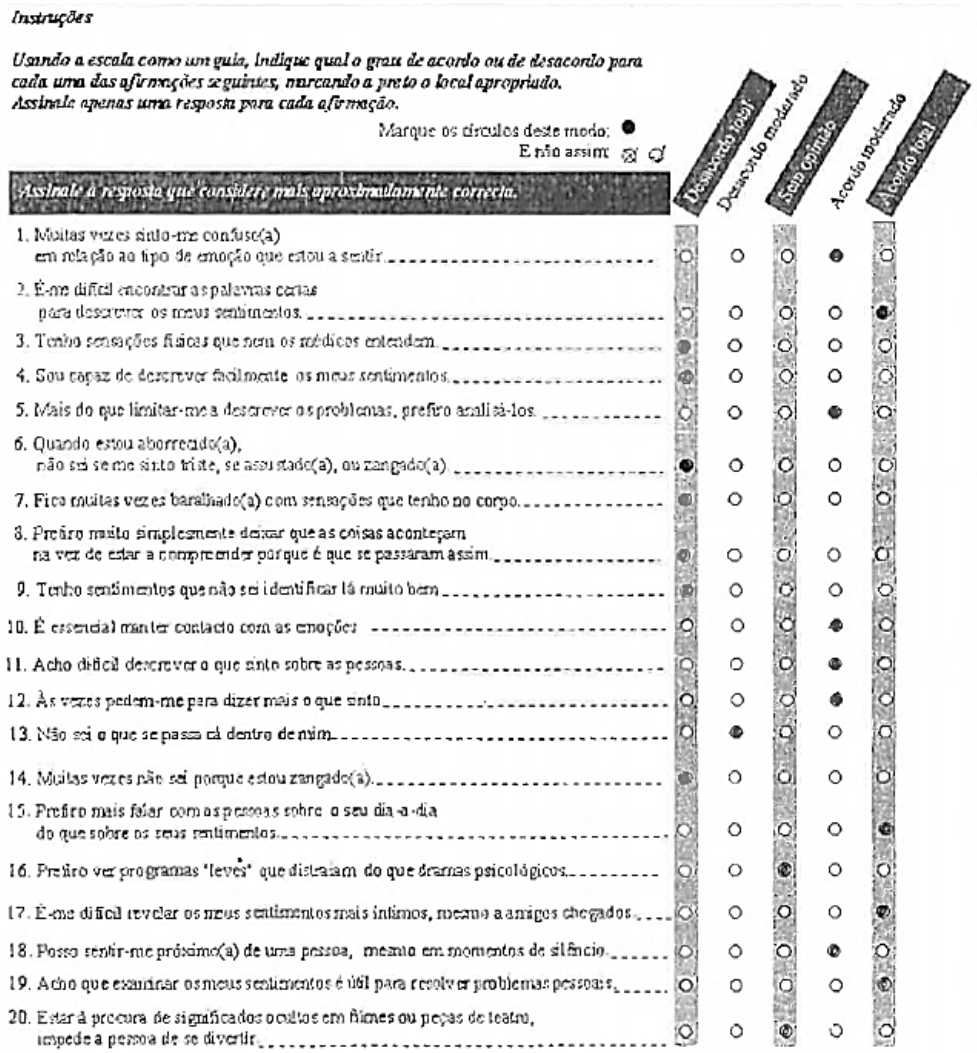
Conclusions

Psychosomatic patients – particularly those with AA – present with complexity and require integrative, multidisciplinary care approaches. These individuals need to be heard and validated, as their clinical presentations often reflect the interplay between somatic and psychological distress.

Prior to receiving such integrative care, many patients undergo numerous medical treatments with limited or transient efficacy. This reinforces a somatic-centric model that neglects the psycho-emotional underpinnings of the disease process, often resulting in delayed or incomplete recovery. Moreover, the repetition of ineffective treatments not only imposes psychological strain but also represents a substantial economic burden on both patients and healthcare systems—further underscoring the need for comprehensive and cost-effective psychosomatic strategies.

The establishment of dedicated psychosomatic care units – and psychodermatology services – within healthcare institutions is of critical importance. Such units would allow for the structured integration of dermatological, psychiatric and psychotherapeutic interventions. Indeed, the presented clinical case clearly illustrates the therapeutic benefits of multidisciplinarity across these domains.

Appendices



Instruções

Usando a escala como um guia, indique qual o grau de acordo ou de desacordo para cada uma das afirmações seguintes, marcando a preto o local apropriado. Assinale apenas uma resposta para cada afirmação.

Marque os círculos deste modo: ● E não assim: ⊘ ◌

Assinale à esquerda a resposta que considerar mais aproximadamente correcta.	Desacordo total	Desacordo moderado	Sem opinião	Acordo moderado	Acordo total
1. Muitas vezes sinto-me confuso(a) em relação ao tipo de emoção que estou a sentir	○	○	○	●	○
2. É-me difícil encontrar as palavras certas para descrever os meus sentimentos	○	○	○	○	●
3. Tenho sensações físicas que nem os médicos entendem	●	○	○	○	○
4. Sou capaz de descrever facilmente os meus sentimentos	●	○	○	○	○
5. Mais do que limitar-me a descrever os problemas, prefiro analisá-los	○	○	○	●	○
6. Quando estou aborrecido(a), não sei se me sinto triste, se assustado(a), ou zangado(a)	●	○	○	○	○
7. Fico muitas vezes baralhado(a) com sensações que tenho no corpo	●	○	○	○	○
8. Prefiro muito simplesmente deixar que as coisas aconteçam na vez de estar a compreender porque é que se passaram assim	●	○	○	○	○
9. Tenho sentimentos que não sei identificar lá muito bem	●	○	○	○	○
10. É essencial manter contacto com as emoções	○	○	○	●	○
11. Acho difícil descrever o que sinto sobre as pessoas	○	○	○	●	○
12. Às vezes pedem-me para dizer mais o que sinto	○	○	○	●	○
13. Não sei o que se passa cá dentro de mim	○	●	○	○	○
14. Muitas vezes não sei porque estou zangado(a)	●	○	○	○	○
15. Prefiro mais falar com as pessoas sobre o seu dia-a-dia do que sobre os seus sentimentos	○	○	○	○	●
16. Prefiro ver programas "leves" que distraiam do que dramas psicológicos	○	○	●	○	○
17. É-me difícil revelar os meus sentimentos mais íntimos, mesmo a amigos chegados	○	○	○	○	●
18. Posso sentir-me próximo(a) de uma pessoa, mesmo em momentos de silêncio	○	○	○	●	○
19. Acho que examinar os meus sentimentos é útil para resolver problemas pessoais	○	○	○	○	●
20. Estar à procura de significados ocultos em filmes ou peças de teatro, impede a pessoa de se divertir	○	○	●	○	○

Figure 1 – 20-item Toronto Alexithymia Scale (TAS-20) of H.

Total score of 62 (alexithymic range): DIF 25 (high) + DDF 16 (high) + EOT 21 (moderate)

Appendices

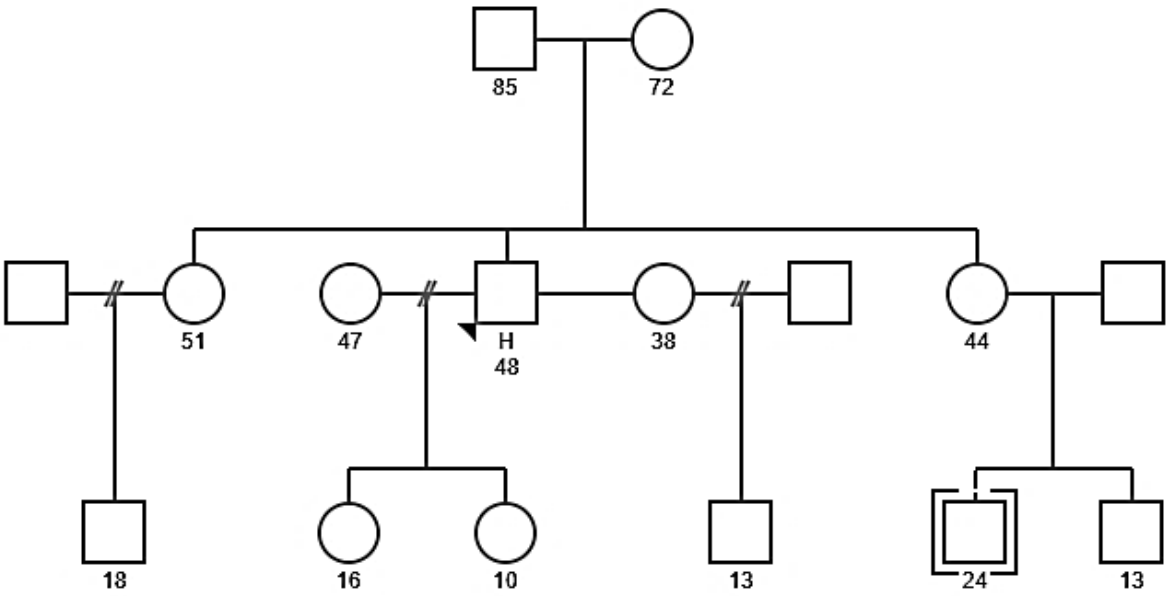
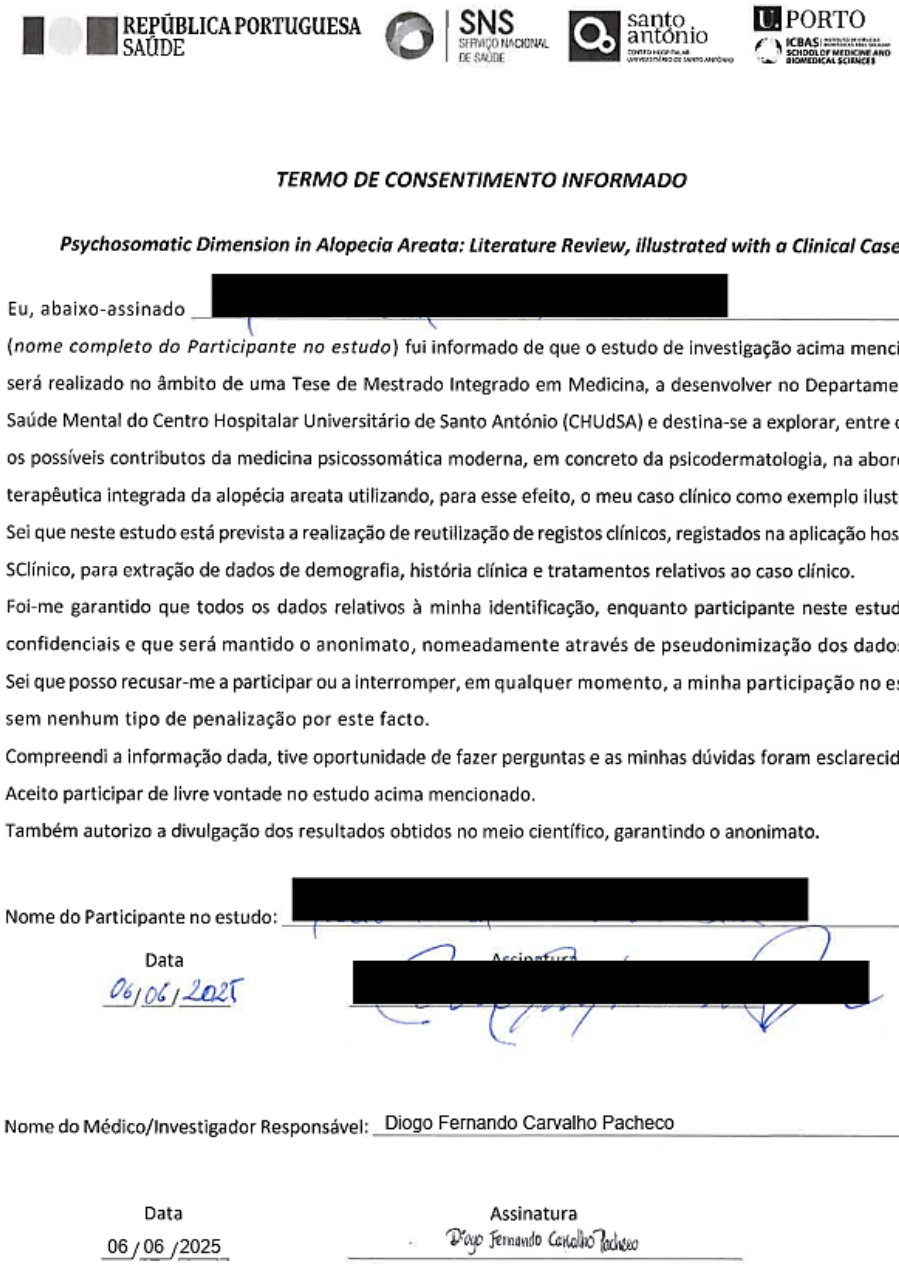


Figure 2 – Three generational genogram of H.

Created using Progeny Genetics software (Progeny Genetics LLC, n.d.).[96]

Appendices



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TERMO DE CONSENTIMENTO INFORMADO

Psychosomatic Dimension in Alopecia Areata: Literature Review, illustrated with a Clinical Case

Eu, abaixo-assinado [redacted]

(*nome completo do Participante no estudo*) fui informado de que o estudo de investigação acima mencionado será realizado no âmbito de uma Tese de Mestrado Integrado em Medicina, a desenvolver no Departamento de Saúde Mental do Centro Hospitalar Universitário de Santo António (CHUdSA) e destina-se a explorar, entre outros, os possíveis contributos da medicina psicossomática moderna, em concreto da psicodermatologia, na abordagem terapêutica integrada da alopécia areata utilizando, para esse efeito, o meu caso clínico como exemplo ilustrativo.

Sei que neste estudo está prevista a realização de reutilização de registos clínicos, registados na aplicação hospitalar SClínico, para extração de dados de demografia, história clínica e tratamentos relativos ao caso clínico.

Foi-me garantido que todos os dados relativos à minha identificação, enquanto participante neste estudo, são confidenciais e que será mantido o anonimato, nomeadamente através de pseudonimização dos dados.

Sei que posso recusar-me a participar ou a interromper, em qualquer momento, a minha participação no estudo, sem nenhum tipo de penalização por este facto.

Compreendi a informação dada, tive oportunidade de fazer perguntas e as minhas dúvidas foram esclarecidas.

Aceito participar de livre vontade no estudo acima mencionado.

Também autorizo a divulgação dos resultados obtidos no meio científico, garantindo o anonimato.

Nome do Participante no estudo: [redacted]

Data 06/06/2025 Assinatura [redacted]

Nome do Médico/Investigador Responsável: Diogo Fernando Carvalho Pacheco

Data 06/06/2025 Assinatura Diogo Fernando Carvalho Pacheco

Figure 3 – Written informed consent assigned by H.

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