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Eating disorders and the skin

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Abstract Eating disorders, which include anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified, are psychiatric disorders with physical complications. Several factors may contribute to the onset of anorexia nervosa and bulimia nervosa, including a familial predisposition to these disorders as well as individual personality characteristics. Dissatisfaction with body shape and an overwhelming desire to be thin are considered as risk factors for the development of eating disorders. Skin signs are the expression of the medical consequences of starvation, vomiting, abuse of drugs, such as laxatives and diuretics, and psychiatric morbidity. They include xerosis, lanugolike body hair, telogen effluvium, carotenoderma, acne, hyperpigmentation, seborrheic dermatitis, acrocyanosis, perniosis, petechiae, livedo reticularis, interdigital intertrigo, paronychia, acquired striae distensae, and acral coldness. The most characteristic cutaneous sign of vomiting is Russell sign (knuckle calluses). Symptoms due to laxative or diuretic abuse include adverse reactions to drugs. Symptoms due to psychiatric morbidity (artefacta) include the consequences of self-induced trauma. The role of the dermatologist in the management of eating disorders is to make an early diagnosis of the "hidden" signs of eating disorders in patients who tend to minimize or deny their disorder.
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Introduction

Eating disorders (EDs) are psychiatric conditions complicated by multiple organ dysfunctions due to malnutrition, binging, purging, and excessive compulsive exercise, potentially leading to a variety of severe, life-threatening medical consequences. EDs include anorexia nervosa (AN), bulimia nervosa (BN), and eating disorder not otherwise specified (ED-NOS). The latest diagnostic criteria for AN and BN, proposed by the American Psychiatric Association, are listed in the version of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision, revised in 2000.[1] Some of the salient clinical features of ED[1] are listed below:

Diagnostic criteria for AN

The diagnostic criteria for AN include[2]:

1. refusal to maintain body weight at or above a minimally normal weight for age and height, including weight loss leading to maintenance of body weight less than 85% of that expected or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected;
2. intense fear of gaining weight or becoming fat, even though underweight;
3. disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight; and
4. in postmenarcheal women, amenorrhea (ie, the absence of at least three consecutive menstrual cycles).

AN is classified according to whether, during the current episode of AN, the patient has not regularly engaged

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("restricting type") or regularly engaged ("binge-eating/purging type") in binge-eating or purging behavior.

Diagnostic criteria for BN

The diagnostic criteria for BN include[2]:

1. recurrent episodes of binge eating, eating is characterized by both of the following:
   - eating, in a discrete period of time (eg, within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances;
   - a sense of lack of control over eating during the episode (eg, a feeling that one cannot stop eating or control what or how much one is eating);
2. recurrent inappropriate compensatory behavior to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise;
3. the binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months;
4. self-evaluation is unduly influenced by body shape and weight; and
5. the disturbance does not occur exclusively during episodes of AN

BN is classified according to whether, during the current episode of BN, the patient is regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas ("purging type"), or the patient uses other compensatory behaviors, such as excessive exercise or fasting ("nonpurging type"), but not regular purging.

Diagnostic criteria for ED-NOS

The ED-NOS category is for disorders of eating that do not meet the criteria for any specific ED. An example would be a woman in whom all of the criteria for AN are met except that the individual has regular menses, or all of the criteria for BN are met except that the binge eating and inappropriate compensatory mechanisms occur at a frequency of less than twice a week or for less than 3 months. ED-NOS also includes binge-eating disorder, which presents as recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviors characteristic of BN.

Epidemiology and risk factors

Most research into the incidence and prevalence of AN has been done in Western countries that are developed and industrialized, because EDs are more widespread in Western culture.[1] These results are generally not applicable outside these areas. EDs are generally considered "culture-bound syndromes," but recent cross-cultural reviews indicate an increase also in non-Western countries. The lifetime prevalence of AN[1] and BN[1] among women is 0.5% and 1% to 3%, respectively, with a higher prevalence of the subthreshold disorders (ie, ED-NOS). In men,[1] the prevalence rates of both AN and BN are about one-tenth the rate for women.

Common risk factors include female sex, ethnicity, early childhood eating problems, childhood gastrointestinal problems, weight and shape concerns, low self-esteem and general negative self-evaluation, sexual abuse, other adverse experiences, and general psychiatric morbidity. AN has been linked to depressive disorders and obsessive-compulsive tendencies such as a preoccupation with thoughts of food. Precipitating risk factors include teasing for being overweight, trying to control body weight and shape for athletic pursuits, feeling anxiety about sexuality, experiencing interpersonal difficulties, and difficulty managing separations.

Skin signs in ED

At least 40 dermatologic signs have been reported in ED patients. Skin signs may lead to the diagnosis of an occult ED.[2-11] Skin signs are due to starvation, self-induced vomiting, drug consumption, and concomitant psychiatric illness (Table 1). Dissatisfaction with the appearance of the skin has been also reported in AN patients.[12]

Stigmata due to starvation include signs on the skin, hair, and nails, and oral cavity. Frequent signs include asteatotic skin and follicular hyperkeratosis, carotenoderma, hyperpigmentation, acrocyanosis and perniosis, acne, and pruritus. Less frequent signs include striae distensae, seborrheic dermatitis, diffuse reticulate purpura, pellagra, scurvy, acquired acrodermatitis enteropathica, and erythema ab igne. Hair and nail changes include lanugolike body hair, alopecia, opaque hair, pili torti, and nail fragility. Oral cavity signs include angular cheilitis, gingivitis, and taste abnormalities.

A body mass index of 16 kg/m^2 or less can be considered a critical value at which skin changes are more frequent. Some signs are characteristic of EDs and have been considered as "guiding signs" (Table 1) to suspect the presence of a still not diagnosed ED. Guiding signs include lanugolike body hair due to starvation, Russell sign, enamel erosions due to self induced-vomiting, and self-induced dermatoses due to psychiatric comorbidity. Guiding signs are not reported in other diseases such as starvation caused by famine, protein energy malnutrition, marasmus, Kwashiorkor, or vitamin C and K deficiencies.

Table 1 Skin signs in eating disorders [a]

Causes	Skin signs
Starvation	**Lanugolike body hair**, asteatotic skin and follicular hyperkeratosis, carotenoderma, hyperpigmentation, acrocyanosis perniosis, acne, pruritus, striae distensae, seborrheic dermatitis, diffuse reticulate purpura, pellagra, scurvy, acquired acrodermatitis enteropathica, erythema ab igne, alopecia, opaque hair, pili torti, nail fragility, angular cheilitis, gingivitis, taste abnormalities
Self-induced vomiting	**Perimylolysis, Russell sign**
Drug consumption	Adverse reactions of drugs, such as laxatives, diuretics and appetite suppressants: photosensitivity, fixed drug eruption, urticaria. Finger clubbing may be due to abuse of laxatives containing senna.
Concomitant psychiatric illness	**Self-mutilation** (eg, skin cutting and burning), trichotillomania, dissatisfaction with the appearance of the skin

[a] Guiding signs in bold type.

Signs due to starvation

Lanugolike body hair

Lanugolike body hair is considered a guiding sign to suspect the presence of AN, because it does not appear when starvation is due to other causes.[2-11] Lanugolike body hair is a frequent sign in AN, especially in younger patients. It presents as fine, downy, pigmented hairs on the back, abdomen, and forearms. It is not a sign of virilization and has been associated with decreased activity of the 5-α-reductase enzyme system, probably due to hypothyroidism.

Asteatosis (xerosis)

Asteatosis (xerosis) is reported in 70% of patients with AN.[2-11] It is caused by a decrease in skin surface lipids, by compensatory hypothyroidism, diuretics abuse, cold intolerance, arm or leg coldness, and compulsive washing. Diminished secretion of sebum usually occurs by the fourth week of starvation, but sometimes it is seen as early as 1 or 2 weeks. Sebum production was found to decrease with an average reduction of 40%. The composition of the sebum was also altered, and this could be detected as early as 5 days after the onset of the fast. The qualitative changes in surface lipids consisted of a reduction in the amount of triglyceride fatty acids, wax esters, cholesterol, and cholesterol esters but with no significant alteration in the amount of squalene.

Carotenoderma and hypercarotenemia

The most characteristic sign of hypercarotenemia is yellow pigmentation in areas of thickened stratum corneum, including the palms, soles, and nasolabial folds when serum levels of carotene exceed 250 μg/dL (threshold <250 μg/dL in children). Despite the striking dermatologic findings, patients usually lack any symptoms. The association between AN and hypercarotenemia has been reported. Several mechanisms have been postulated but none confirmed.

Carotenemia in AN is chiefly related to a diet that is rich in β-carotene sources, mainly carrots and other low-energy yellow vegetables. Some consider the cause to be an acquired defect in the metabolism or utilization of vitamin A. It may also be related to abnormalities of lipid metabolism, such as the decreased catabolism of β-lipoprotein and hypothyroidism, which are observed in this disease.[2-11,13]

Acrocyanosis, pernio

Acrocyanosis is a disorder of the peripheral circulation characterized by cyanosis and coldness of the hands and feet. The pathophysiologic changes consist of constriction of the skin arterioles and venous dilatation. The onset is most commonly during adolescence and early adult life, and the ratio of female-to-male cases is in the order of 6:1. This sex difference may be partly explained by the prevalence of acrocyanosis in patients suffering from AN, a disorder that is seen predominantly in teenaged girls and young women.

Why acrocyanosis should occur in AN is still uncertain. It appears to be more prevalent among the more severely ill patients.[14-16] Acrocyanosis also appears to be associated with facial and truncal pallor, a slower pulse rate, and higher fasting plasma glucose levels. It may be a protective mechanism against excessive heat loss, dehydration, emotional strain, or a primary disturbance in thermoregulation. Hand blood flow is reduced in AN, although the mean skin temperature over a number of sites is comparable to that in healthy controls. A study of rectal and skin temperature and tissue heat conductance before and after exercise has also revealed evidence of increased vasoconstriction in the limbs of individuals with AN.

The association of acrocyanosis with evidence of increased vasoconstriction in the skin of the face and trunk is consistent with this view, as is the predilection for the more severe cases. Among adolescents, pernio has been seen in association with AN. All patients were very thin, and thin body habitus may be associated with increased cutaneous vasoreactivity.

Acne in AN

Acne is reported in 47% to 59%.[2-7] The specific occurrence of the onset of acne in some patients only at the time of weight gain and at a weight when they were

previously not affected by acne suggests a mechanism related to the re-establishment of physiologic homeostasis, especially relating to endocrine function. Particularly attractive is the hypothesis of hormonal changes associated with weight gain triggering the development of acne. A number of abnormalities in AN reflect endocrine disturbances, and specifically, sex hormone production and metabolism are altered as a consequence of a defect in the hypothalamic-pituitary-gonadal axis. These abnormalities are reversible upon improved nutrition.

I was not, however, able to demonstrate a relationship between acne and androgen status both clinical and biochemical parameters. It is, of course, possible that the particular androgens assayed do not reflect those responsible for the development of acne in postpubertal women, who were either previously unaffected by acne or had long recovered from adolescent skin problems. With the recent resurgence of interest in zinc in the pathogenesis of various skin disorders, specifically acne, some authors examined those patients with acne in an effort to detect any zinc deficiency. Although levels were low in some, there was no difference between the women who were and were not affected by acne.

Polycystic ovarian syndrome in BN

An interesting question is what is the etiologic link between BN and clinical manifestations of polycystic ovarian syndrome.[16] Polycystic ovarian morphology may be secondary to abnormal eating behavior. Another explanation for an association between BN and polycystic ovarian syndrome may be that hyperandrogenism is a primary condition that predisposes for the development of bulimic behavior. A subset of bulimic individuals may have a primary endocrine/metabolic disorder that may promote bulimic behavior because androgens have appetite-stimulating effects and could impair impulse control. Preliminary reports suggest that medication with antiandrogens may have beneficial effects on some of the symptoms of bulimia.

Pruritus

A statistically significant association between pruritus and AN, with greater severity at low weight and resolution with weight restoration, has been reported.[17-19] Changes in BMI correlate with a change in itch severity, with resolution of itching on weight restoration. The apparent association between AN and pruritus has several possible explanations.

Pruritus may be a product of the patients' changing psychopathology. Mental state characteristics are known to modulate sensory perception through gating of spinal pathways by descending Raphe fibers, although anxiety and dysphoria are more commonly associated with weight restoration, which would predict worsening pruritus on weight gain. Endocrine factors are pertinent. Lowered serum androgens would reduce sebaceous gland activity, which is known to be associated with caloric deprivation.

In some patients with AN, the common findings of impaired renal, hepatic, and thyroid function might manifest as pruritus, but this was only true for thyroid function in a sample and only in a minority of subjects. Their contributory role cannot be excluded, however. The simplest explanation is that pruritus results from eczema in anorexia, with dermatitis occurring in just under half the sample and resolving on weight restoration in four of five cases. This is a plausible explanation for a minority of cases, and future studies might assess dermatitis empirically, with the option of treatment intervention and analysis of the contribution of nutritional deficits in anorexia, such as zinc deficiency. Alternatively, AN has been associated with altered thresholds for thermoregulation and vasodilatation. Compulsive behaviors, such as repeated washing, are established features of AN and may worsen pruritus.

Striae distensae

Studies have reported striae distensae only in men with AN.[7,8] Cortisol is increased in AN, but the relationship of striae distensae with excess cortisol production is controversial.

Purpura

The more frequent changes in peripheral blood cell count are leukopenia and anemia, whereas thrombocytopenia is reported less frequently. Fifty percent of patients with blood parameters within normal reference ranges showed signs of bone marrow atrophy, so that some authors concluded that blood parameters within normal reference ranges are not reliable predictors of bone marrow impairment. These abnormalities are not generally due to iron, vitamin B_{12}, or folic deficiencies. In fact, anemia is generally normochromic and normocytic with a normal or high level of iron and ferritin: anorectic girls save their iron stores more than healthy girls because of the absence of menstrual cycle; nevertheless, iron deficiency due to malnutrition may also be present, and if relevant, it can cause microcytic anemia. The degree of cellular loss may range from a mild hypoplasia, with a relative increase in fat tissue, to a gelatinous transformation of the bone marrow with fat cell atrophy in the most severe cases.[20-22]

Diffuse reticulate purpura

A diffuse reticulate purpura has been reported in AN.[23] The eruption may be the result of bone marrow depression from starvation and the subsequent thrombocytopenia. This hypothesis, unfortunately, seems unable to explain in full the entire clinical picture. Loosened capillary walls and weakened dermal supporting structure induced by severe malnutrition and rapid weight loss may be the most probable

contributing factors of reticulate purpura. This form is a rare, unique, and characteristic cutaneous manifestation of AN.

Acrodermatitis

The most common nutritional deficiencies causing a periorificial or acrodermatitis are zinc deficiency, biotin deficiency, kwashiorkor, and essential fatty acid deficiency. Whereas older literature focused on the relationship among nutritional deficiency, malnutrition, and poverty, recent research has identified additional patient populations that are at risk for developing nutritional deficiencies include AN.[24]

Alopecia, hair loss, and opaque and fragile hair

Alopecia, hair loss, and opaque and fragile hair is reported in 17% to 61% of patients.[2-11] It has a diffuse pattern, sometimes with a frontal predominance. Opaque and fragile hair (as in protein deficiency or crash diets) is reported. There is an increased number of telogen hairs. A link between excessive vitamin A intake and hair loss is well established. Hypothyroid state is a predisposing factor.

Pili torti

Pili torti are characterized by a twisting of the hair shaft on its own axis. Pili torti may occur as an inherited, isolated phenomenon with the onset at birth or in the early months of life. They have been reported in AN.[25] No true pili torti were found in a series of 30 patients with severe AN, but twisted hair were found in 6.6%.[26] A literature review shows that the largest series of pili torti (congenital and acquired) have been published by authors from Middle Eastern countries, such as authors from Israel and Egypt. We, therefore, hypothesized that, under the same conditions, a genetic factor may predispose to this hair shaft defect.[26]

Nails

The following frequencies of nail abnormalities have been reported in AN[2-11]: onychoschizia, 9%; fragility, 15% to 33%; longitudinal striae, 15%; and pitting and periungual erythema 20% to 48%.

Oral cavity

Angular cheilitis, gingivitis, and enamel erosions may be due to starvation.[27] The usual disturbed anorectic diet is an important factor affecting teeth. Anorectic patients prefer "slimming" foods, in particular, raw citrus fruits, such as lemons and grapefruits, or their juices. In some cases, fruit was eaten each day over a period of months or years, and in those cases where it was done specifically to induce diarrhea, it completely replaced a normal diet. The pH value of these substances is 3.5, a concentration at which enamel decalcification occurs. As a consequence of starvation, which induces wasting and dehydration, secretions are reduced. The quantity and composition of saliva is affected, and this alteration caused by electrolyte imbalance may lower the buffering and remineralizing capacity of the saliva, making the teeth more susceptible to acid attack. In patients who practice vomiting, the saliva has a reduced pH value and leads to dental erosions.

In bulimic patients, marked bilateral salivary gland enlargement is reported. It is likely that the parotid gland enlargement is sialadenosis, a diagnosis applied to diffuse, asymptomatic, noninflammatory salivary gland enlargement.

Skin signs due to vomiting

The most characteristic cutaneous sign due to self-induced vomiting in purging-type AN is the Russell sign (knuckle calluses). The lesions involve calluses on the dorsal aspects of the dominant hand induced by the patient's repeated introduction of the hand into the mouth. It is a guiding sign in the diagnosis of eating disorders.[28]

Skin signs due to laxative or diuretic abuse

In purging type AN, the anorectic patient may undergo the adverse reactions of drugs, such as laxatives, diuretics, and appetite suppressants, which they use. Thiazide diuretics, for example, may induce photosensitivity; phenothiazines, fixed drug eruption, and phenolphthalein laxatives, urticaria. Finger clubbing may be due to abuse of laxatives containing senna.[29]

Skin signs due to concomitant psychiatric illness

Patients with ED are at high risk for self-mutilation, including skin cutting and burning. The self-mutilation in these patients is regarded as a borderline personality disorder symptom. The combination of self-mutilation, anorexia, bulimia, and other symptoms, such as episodic alcohol abuse and swallowing foreign objects, may be manifestations of an impulse control disorder known as the "deliberate self-harm syndrome."[30] Artifact scars as a visible result of this behavior are reported in 13% of adults with AN and in 30% of children and adolescents.

Trichotillomania has also been reported and is more prevalent in bulimic than in nonbulimic females.

Dissatisfaction with the appearance of the skin

The body image disturbance in ED is not necessarily confined to a distorted perception of body weight and shape but may generalize to concerns regarding other aspects of body image, including dissatisfaction with skin

appearance.[12] The attributes that were rated most frequently by women with EDs are associated with aging of the appearance such as bags under the eyes, darkness under the eyes, dryness of the skin, freckles, fine wrinkles, patchy hyperpigmentation, and roughness of the skin. A major psychosocial factor that is believed to contribute to the development of eating disorders is the patient's difficulties in dealing with the developmental tasks of adulthood and a tendency to want to retreat to the security of the preadolescent years. It is possible that a greater concern about the skin changes associated with aging among the women with eating disorders is an index of this developmental conflict.

Prognosis of skin signs in ED

Skin signs disappear when the patient gains weight. The resolution of skin eruptions in patients with ED almost always depends on the treatment of the underlying disorder.[31]

Conclusions

EDs are psychiatric pathologies with medical implications that require a multidisciplinary team approach. Much attention must be paid to the identification of early signs associated with the development of EDs because the prognosis is better if the diagnosis and therapy are timely. Dermatologists have an important role in the early diagnosis of ED because skin signs are, at times, the only easily detectable symptoms of hidden AN and BN.[32] Forty cutaneous signs have been recognized, and new reports are expected owing to the increasing frequency of this pathology all around the world.

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