The etiology, diagnosis, and management of hyperhidrosis: A comprehensive review

Etiology and clinical work-up

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Learning objectives
After completing this learning activity, participants should be able to discuss the etiology of hyperhidrosis; describe the prevalence and demographical characteristics of hyperhidrosis; explain the diagnostic work-up of hyperhidrosis; recognize the burden and psychosocial effects of hyperhidrosis; list resources available for hyperhidrosis patients; and assess the validity and reliability of existing studies related to hyperhidrosis.

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Hyperhidrosis (HH) is a dermatologic disorder defined by sweat production exceeding thermoregulatory needs. Clinically, HH is diagnosed when excess sweating creates significant emotional, physical, or social discomfort, causing a negative impact on the patient's quality of life. Existing data imply that this condition may affect at least 4.8% of the US population. The etiology of HH may stem from a complex autonomic nervous system dysfunction, resulting in neurogenic overactivity of otherwise normal eccrine sweat glands. Alternatively, HH may be a result of aberrant central control of emotions. This condition is categorized as primary or secondary HH. Approximately 93% of patients with HH have primary HH, of whom >90% have a typical focal and bilateral distribution affecting the axillae, palms, soles, and craniofacial areas. Secondary HH presents in a more generalized and asymmetric distribution and is generated by various underlying diseases or medications. Secondary causes of HH need to be excluded before diagnosing primary HH. (J Am Acad Dermatol 2019;81:657-66.)

Key words: apocrine sweat gland; apoeccrine sweat gland; axillary sweating; autonomic nervous system; eccrine sweat gland; emotional sweating; excessive sweating; facial sweating; hyperhidrosis; oversweating; palmar sweating; plantar sweating; primary hyperhidrosis; secondary hyperhidrosis; sweat overproduction; thermoregulatory sweating.

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EPIDEMIOLOGY

Key points

- **Hyperhidrosis is a dermatologic disorder characterized by excessive sweat production.**
- **Existing data imply that this condition may affect at least 4.8% of the US population**
- **Primary hyperhidrosis constitutes 93% of all cases of hyperhidrosis**
- **The onset of primary hyperhidrosis typically occurs between 14 and 25 years of age**
- **Common locations affected by primary hyperhidrosis include the axillae, palms, soles of the feet, and craniofacial areas**

Hyperhidrosis (HH) is characterized by excessive sweat production. The prevalence of HH is difficult to determine because of the embarrassing nature of its presentation, the lack of awareness concerning its medical nature, therapeutic options that deter many patients from reporting their symptoms, and the inconsistent methodologies used in HH epidemiologic studies.[1] Early studies reported a prevalence rate of 0.6% to 1% in the general population.[2] A 2004 survey determined that approximately 2.8% of Americans have HH, of whom 50.8% have axillary HH.[3] Only 38% reported discussing their symptoms with a health care professional (47.5% of females and 28.6% of males).[3] A more recent 2016 online survey with a nominal response rate of 4.5% suggested that approximately 4.8% of the US population, or 15.3 million individuals, have HH.[1] The prevalence of HH for the US population is far below the reported prevalence in other countries,[4-7] and therefore it is speculated that the cited finding of 4.8% is an underestimation. A 2011 review indicated that 93% of patients with HH had primary HH. More than 90% of patients with primary HH experienced symptoms involving the following commonly affected locations: axillae, palms, soles, and craniofacial regions.[8,9]

Both males and females are equally affected,[3] although females more frequently seek treatment than males[10,11] and are further inclined to complain of axillary HH.[11] Males are more likely to experience craniofacial HH and to complain of additional affected areas.[9,12]

HH can present at any age.[13] The average age of onset for primary HH ranges from 14 to 25 years of age.[14] The prevalence of HH is higher among individuals 18 to 39 years of age than among adults >65 years of age and children <18 years of age.[1] The lower prevalence of the disease in the advanced age population may represent spontaneous regression of HH later in life.[9,13] When HH presents before puberty, the disorder most commonly affects the plantar or palmar regions (88.9%) and less frequently involves the axilla (15.5%), face (6.6%), or dorsal and abdominal regions (4.4%).[9,15] After puberty, onset is more commonly associated with an axillary distribution.[3,9,12,16] The overall distribution of commonly involved anatomic sites affected by HH is as follows: axillae 51%, plantar 30%, palmar 24%, and facial 10%.[3,17] Of affected patients with HH, 18% have simultaneous axillary and palmar expressions, whereas 15% have concurrent palmar and plantar manifestations.[3,17] Less commonly affected areas include the trunk (3%) and inguinal folds (1.3%). Other uncommon locations, each affecting <1% of patients with HH include the buttocks, neck, wrists, legs, and submammary regions.[8,9]

ANATOMY AND MECHANISM OF SWEATING

Key points

- **Three types of sweat glands exist: eccrine, apocrine, and apoeccrine**
- **Eccrine sweat glands are the most abundant type of sweat glands**
- **The autonomic nervous system regulates sweat production**

Thermal regulation involves the dissipation of body heat mainly via the evaporation of sweat, as well as evaporation of water from the respiratory tract.[18] Embryologically, sweat glands develop from an epidermal surface ingrowth that evolves into ducts and glands with differentiated cells.[19,20] Three types of sweat glands exist: eccrine, apocrine, and apoeccrine.

Eccrine sweat glands are the most numerous among the 3 types of sweat glands and form only during the embryonic stage of development.[21,22] Approximately 3 million eccrine sweat glands are unevenly distributed over the entire body surface area.[23] Eccrine sweat glands are particularly abundant in the palms, soles, forehead, axillae, and cheeks, and are less numerous on the back and chest.[20,23-26] Eccrine sweat glands are the only sweat glands present on the palms.[13] They do not usually exist on the mucosal lips, external auditory canal, nipples, glans penis, clitoris, labia minora, or nail beds.[20] Eccrine sweat glands are comprised of a dermal secretory tubule, an intraepidermal duct, and an intraepidermal pore that opens onto the skin surface,[27] and produce an odorless, clear, thin hypotonic secretion.[27,28] The rate and volume of eccrine sweat production fluctuate and are based upon thermoregulatory needs.[23] Emotional, gustatory, or physical stimuli can increase the secretion

rates up to 10 L/day.[13,27,29] Eccrine sweat glands are innervated by sympathetic cholinergic nerve fibers.[30] Eccrine glands also respond to catecholamines in emotionally induced sweating.[31,32] The normal secretion rate of eccrine sweat glands is 0.5 to 1 mL/minute,[23] and only 5% of glands secrete sweat at any given time.[20] In severe HH, secretion may exceed 40 mL/m^2/minute.[23,24] Eccrine sweat glands are believed to be responsible for primary HH.[13,24,33]

Apocrine sweat glands are far less numerous than eccrine sweat glands. The number and size of apocrine sweat glands increase until 18 years of age, and they become active during puberty.[18,34] Compared to eccrine glands, the apocrine sweat glands are larger, located deeper within the dermis, contain secretory coils that can form a tubular network, have a larger sac-shaped secretory tubule lumen, and are comprised of different epithelial cell compositions. Apocrine sweat glands release sweat into the infundibular portion of the hair follicle rather than straight onto the skin surface.[13] Apocrine sweat glands are restricted to the axillary, anogenital, perimammary (mammary glands), periumbilical, preputial, scrotal, eyelid (Moll glands), and external auditory conduit (ceruminous glands) areas.[35,36] The ratio of apocrine to eccrine sweat glands is 1:1 in the axillae and 1:10 in other areas.[37] Apocrine sweat glands produce viscid and odorous sweat,[29,38] with their secretion comprising proteins, fatty acids, sugars, ammonia, and occasionally chromogens.[29,36,38] Apocrine sweat gland function in humans remains poorly understood, but may involve the production of pheromones and body odor.[13] Apocrine sweat glands are under the control of adrenergic nerve fibers.[14,26,31,32,35,39] The role of apocrine sweat glands in HH (mainly axillary) is assumed to be insignificant.[29]

The existence of apoeccrine sweat glands remains controversial.[31] They are thought to differentiate and develop from eccrine sweat glands[23,37] after adolescence[40] and to produce copious, watery secretions.[23] These glands possess morphologic characteristics shared by both eccrine and apocrine sweat glands.[40] Apoeccrine sweat glands are mainly identified in the axillary and perianal locations and constitute approximately 10% to 45% of the entire axillary sweat glands.[23,36] They are sympathetically innervated and are sensitive to both adrenergic and cholinergic stimuli.[13,41,42] Apoeccrine sweat gland secretion can be 7 times higher than that of eccrine sweat glands,[42] potentially implicating their role in the pathophysiology of axillary HH.[13]

Thermoregulation, and therefore sweat production, is regulated by the cerebral cortical structures, the preoptic region of the anterior hypothalamus,[43] and the sympathetic nervous system.[44] Thermal receptors are dispersed throughout the body and are found in internal organs, the hypothalamus,[45] the brain stem,[46] the spinal cord,[47] and the skin. Afferent nerve fibers carry signals to the hypothalamus through the lateral spinal cord. The efferent sympathetic sudomotor pathway for thermoregulation runs as follows: from the cerebral cortex to the hypothalamus; hypothalamus to medulla; fibers cross in the medulla oblongata and continue to the lateral horn of the spinal cord; lateral horn to the intermediolateral cell nuclei of the spinal cord paravertebral sympathetic ganglia; and unmyelinated, postganglionic sympathetic C class type nerve fibers stimulate the eccrine sweat gland postsynaptic muscarinic receptors (Fig 1).[20,23,45,48-50]

PATHOPHYSIOLOGY

Key points

- **Primary hyperhidrosis results from the excessive neurogenic activity of otherwise normal eccrine sweat glands**
- **There is no change in the size, number, or histologic appearance of sweat glands in hyperhidrotic patients**
- **Hypothesized etiologies of primary hyperhidrosis include complex autonomic nervous system dysfunction and abnormal central control of emotions**

The watery nature of sweat produced in HH and the sole presence of eccrine sweat glands in the palms both contribute to the theory that eccrine sweat glands are the source of oversecretion in HH.[13,51]

The size or number of eccrine glands in patients with primary HH is not increased,[20,25,52,53] and these sweat glands do not possess any microscopic or macroscopic histopathologic abnormalities.[25,54] Therefore it has been hypothesized that the etiology of HH may stem from a complicated malfunctioning of the autonomic nervous system involving both the sympathetic and parasympathetic systems,[23] causing neurogenic hyperexcitability of the reflex circuits, which leads to hyperstimulation of otherwise normal eccrine sweat glands (and possibly apoeccrine sweat glands).[13,18,23,25,52,55] Enhanced sympathetic sudomotor skin responses were observed in patients with HH, suggesting a regulatory dysfunction.[56-58] In addition, an electroencephalographic analysis of patients with HH showed hyperperfusion of the frontal cortical areas during sweating episodes.[59] In addition, studies on patients with palmar HH have reported differences in cardiac autonomic function, including reduced reflex bradycardia after the

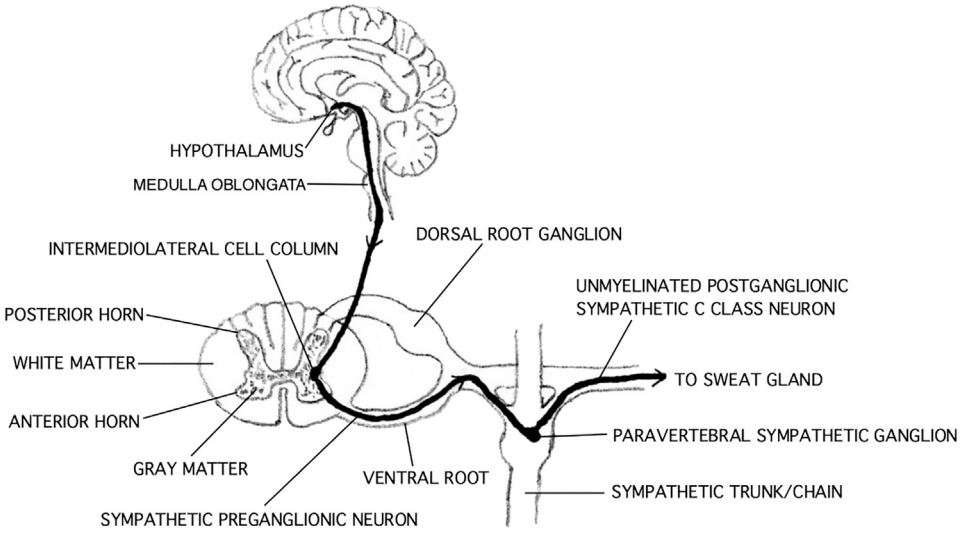

Fig 1. Efferent sympathetic sudomotor pathway for thermoregulation.

Valsalva maneuver and increased vasoconstriction after cold-water finger immersion compared with control subjects.[56] These findings may implicate focal HH as a constituent of a more complex autonomic dysfunction.[60,61]

Another theory postulates that HH is the result of aberrant central control of emotions.[62] Both emotional sweating and thermoregulatory sweating are triggered by sympathetic cholinergic nerves. Emotional sweating is carefully regulated through the limbic system,[31,32] anterior cingulate cortex, and hypothalamus. The areas that control emotional sweating mainly affect the axillae, palms, soles, forehead, and scalp.[63] These areas correspond to commonly affected areas implicated in HH. It is postulated that in patients with HH, the sweat center in the hypothalamus—which regulates perspiration on the palms, soles, and the axillae—is different from other hypothalamic sweat centers in that it is exclusively under the influence of the cortex, without any input from the thermoregulatory components.[35] HH may therefore be driven by an abnormal central control of emotions.[62,64]

CLASSIFICATION OF HYPERHIDROSIS AND DIFFERENTIAL DIAGNOSIS

Key points

- **Primary hyperhidrosis is idiopathic and typically has a focal, bilateral, and symmetric distribution of excessive sweat production**
- **Secondary hyperhidrosis is related to an underlying cause**
- **Generalized primary hyperhidrosis**

HH is classified as a primary or secondary disorder.[8] With regard to anatomic distribution, HH can further be classified into focal, regional, symmetric, asymmetric, or generalized.[20,65]

Primary HH is idiopathic focal, bilateral, and symmetrical exaggerated perspiration that typically affects the axillae, palms, soles, and craniofacial regions[3,66] and is not caused by any underlying medical diseases or medications.[3] Primary HH is diagnosed in 93% of all patients with HH.[8] It can appear continuously or in phases and does not usually occur at night. Primary HH can be induced by thermal provocations, emotional triggers, or physical activity.[18]

Secondary HH is less common than primary HH and is most frequently related to an underlying cause (Fig 2). Secondary HH is most commonly classified as generalized but can also present focally or regionally. Secondary HH occurs when the patient is awake or asleep. Generalized secondary HH may be caused by physiologic conditions, such as excessive heat, fever, pregnancy, or menopause. Potential pathologic causes for secondary HH can include malignancy (lymphoma and other myeloproliferative disorders), infection (acute viral or bacterial infections and chronic infections, including tuberculosis, malaria, brucellosis, or HIV), endocrine/metabolic disorders (diabetes mellitus/hypoglycemia, diabetes insipidus, hyperthyroidism, thyrotoxicosis, pheochromocytoma, acromegaly, hyperpituitarism,

Examples of Secondary Causes of Hyperhidrosis		
Generalized Secondary Hyperhidrosis		
Physiologic		• Heat • Fever • Pregnancy • Menopause
Pathologic	*Malignancy*	• Lymphoma • Myeloproliferative disorder
	Infection	• Acute viral or bacterial infections • Tuberculosis • Malaria • Brucellosis • HIV
	Endocrine/Metabolic	• Diabetes mellitus/hypoglycemia • Diabetes insipidus • Hyperthyroidism • Thyrotoxicosis • Pheochromocytoma • Acromegaly • Hyperpituitarism • Carcinoid syndrome
	Cardiovascular	• Endocarditis • Congestive heart failure • Cardiovascular shock
	Respiratory	• Respiratory failure
	Neurologic	• Stroke • Parkinson's disease • Psychiatric disorders
	Drugs	• Antidepressants (tricyclic antidepressants, anxiolytics, antipsychotics, selective serotonin reuptake inhibitors) • Antibiotics (ciprofloxacin) • Antivirals (acyclovir) • Hypoglycemic agents (insulin, glyburide) • Triptans • Antipyretics • Nonsteroidal anti-inflammatory drugs • Antiemetics • Adrenergic or cholinergic agents • Alcohol • Cocaine • Heroin
Symmetric Focal Secondary Hyperhidrosis		
		• Compensatory Sweating • Physiologic/pathologic gustatory
Asymmetric Focal Secondary Hyperhidrosis		
		• Focal neurologic lesion • Tumors • Eccrine nevus • Frey syndrome
Regional Secondary Hyperhidrosis		
		• Neoplasm • Peripheral neuropathies • Spinal cord lesion • Stroke

Fig 2. Secondary causes of hyperhidrosis.

or carcinoid syndrome), cardiovascular disease (endocarditis, congestive heart failure, or cardiovascular shock), respiratory disease (respiratory failure), neurologic disorders (stroke or Parkinson disease), or psychiatric disorders,[67,68] among others.[8,52,64,69-72] In addition, many drugs can cause secondary generalized HH, including antidepressants (tricyclic antidepressants, anxiolytics, antipsychotics, or selective serotonin reuptake inhibitors), antibiotics (ciprofloxacin), antivirals (acyclovir), hypoglycemic agents (insulin or glyburide), triptans, antipyretics, nonsteroidal antiinflammatory drugs, antiemetics, adrenergic or cholinergic agents, alcohol, cocaine, heroin (including withdrawal), and many others.

Focal or regional secondary HH is rare and can be either symmetric or asymmetric. Symmetric focal secondary HH includes compensatory sweating, an iatrogenic condition caused by surgical sympathectomy for the treatment of primary focal HH, and gustatory sweating.[73] Physiologic gustatory sweating presents as bilateral facial perspiration caused by the consumption of hot or spicy foods and liquids or exposure to heat. Pathologic gustatory sweating can be induced by sympathetic nerve damage from a neoplasm or sympathectomy, postsurgical complication of parotidectomy, auriculotemporal nerve syndrome, diabetic neuropathy, or infection—frequently herpes zoster infection.[9,73-75]

Asymmetric focal secondary HH can be seen in individuals with focal neurologic lesions, tumors, or a variety of cutaneous disorders, including eccrine nevus and Frey syndrome.[76,77] Frey syndrome is a focal facial perspiring neurologic disorder. It is caused by abnormal regeneration of an injured facial nerve due to a parotid tumor or complication of parotidectomy. It is manifested by unilateral facial flushing and sweating while consuming certain foods.[9,74,75]

Secondary regional HH can be caused by a neoplasm, peripheral neuropathy, spinal cord lesion, or stroke.[9,78,79]

Genetics

Of patients with primary HH, 35% to 56% have a positive family history, suggesting a possible genetic association.[3,11,12,80] Research suggests that primary focal HH may be a genetic disorder with variable phenotype, autosomal dominant transmission, and incomplete penetrance.[16,81-86] Complex inheritance with multigene involvement has also been proposed to explain the varying degrees of severity and inconsistent pattern of transmission often observed in families.[16,81] Two loci for primary HH were mapped to chromosomes 2q31.1[82] and 14q11.2-q13,[87] implying locus heterogeneity for this condition.

DIAGNOSIS

Key points

- **Secondary hyperhidrosis needs to be excluded before diagnosing primary hyperhidrosis**
- **Laboratory diagnostic tests are not necessary when primary hyperhidrosis is suspected**
- **In contrast to primary hyperhidrosis, secondary hyperhidrosis is asymmetric, unilateral, or generalized**
- **Secondary hyperhidrosis commonly begins after 25 years of age, lacks a family history, and often involves nocturnal sweating**

Secondary causes of HH need to be excluded before primary HH can be diagnosed.[3,8,44,64] Patient history is commonly sufficient to differentiate between primary HH and secondary underlying causes of oversweating.[65] General questions should focus on the pattern of sweating, age of onset, known initiating causes, duration, frequency, amount, distribution, night sweating, family history, and any symptoms that point to a secondary cause.[27] Related symptoms, such as weight loss, fever, or lymphadenopathy, raise the suspicion of secondary causes.[20] The clinical examination should focus on finding evidence of excessive sweating and any symptoms that point to an underlying secondary cause.[28] Laboratory diagnostic tests are typically not needed for the diagnosis of common localized idiopathic HH.[8,27,30,88] However, based on the history and physical examinations, specific laboratory diagnostic tests need to be obtained if secondary underlying causes are suspected.[20]

Diagnostic principles for the recognition of primary HH have been proposed by the multispecialty working group consensus panel. Specific diagnostic criteria consist of 6 months of visible focal sweating that exceeds thermoregulatory needs and at least two[44] or four[8] of the following, with the latter having an increased specificity and positive predictive value[8]: mainly affecting eccrine-dense areas (the palms, soles, axillae, or craniofacial areas), in a symmetrical bilateral distribution, disturbing daily activities, occurring >1 time per week, having an onset before 25 years of age, having a positive family history, and lacking nocturnal symptoms.[8,44,89,90] Truly generalized primary HH is rare.[9]

Findings that raise the suspicion of secondary HH include an asymmetric, unilateral, or generalized distribution, nocturnal symptoms, onset after 25 years of age, and a negative family history.[8]

SWEAT PRODUCTION TESTS

Key points

- **Various quantitative and qualitative tests for hyperhidrosis exist**
- **Hyperhidrosis testing is not commonly used for clinical purposes**
- **Hyperhidrosis tests can help determine sweating severity and direct therapy**

Quantitative and qualitative methods for examining sweat production are not commonly performed during a clinical examination. However, they can help determine HH severity, guide treatment options, and gather data for clinical research.[20]

Quantitative sweat production tests

The minor starch-iodine test presents an approximate, qualitative estimate of the produced sweat volume, with a useful map of regions affected by sweating before the initiation of treatment. Dusted cornstarch and iodine react with sweat to produce a purplish sediment that identifies affected areas.[27,91,92]

The ninhydrin test involves the reaction of ninhydrin with amino acids present in sweat to produce a vivid color that can be quantified via digital analysis.[20,93]

Gravimetric testing involves weighing sweat produced in a specific area over a fixed period of time using preweighed filter paper.[27,94,95]

The thermoregulatory sweat test involves applying alizarin red, corn starch, and sodium carbonate to the skin to identify hyperhidrotic areas. The patient is then placed in a heated chamber where normal thermoregulatory sweating is induced. In this manner, both hyperhidrotic and thermoregulatory sweating are delineated.

Dynamic quantitative sudometry measures sweating over time. Absorbed moisture is quantified by a chamber placed on the skin surface, through which dried gas is transmitted.[96-98]

The electronic moisture meter is used to detect moisture evaporation from the skin directly.[99]

Qualitative methods for the evaluation of quality of life in patients with HH

The Hyperhidrosis Disease Severity Scale (HDSS)[3,30,100] is an HH-specific questionnaire by which patients rank the influence of sweating on everyday activities on a scale of 4 grades.[3] Scores of 3 and 4 indicate severe HH, a score of 2 indicates moderate HH, and a score of 1 indicates the absence of HH. The Canadian Hyperhidrosis Advisory Committee recommends using this test to direct treatment based on disease severity.[30] Treatment success is defined by an HDSS score change from 4 or 3 to 2 or 1 scores, or a score change from 2 to 1. Treatment failure is defined as lack of improvement in HDSS score after 1 month of treatment or intolerable therapy. A 1-point HDSS score improvement has been shown to correlate with a 50% sweat reduction. An improvement of 2 points corresponds to an 80% sweat reduction.[30]

The Hyperhidrosis Impact Questionnaire[20,91,101,102] assesses the impact of HH on daily life, as well as treatment efficacy. It consists of a 41-item module baseline questionnaire and 10-item assessment follow-up questionnaire.

The dermatology life quality index is used for a variety of dermatologic conditions to measure both the effect of the disorder on quality of life and the improvement in quality of life after treatment.[103,104] It is a self-conducted analysis including 10 questions rating 10 different domains.[91,103,105]

The Medical Outcomes Trust Short Form 12 Health Survey[20,101,106] is a quality of life questionnaire with a 12-item survey assessing patients' own views of general health, emotional health, social functioning, and physical activity.

Complications

The moist environment induced by HH increases the risk of cutaneous bacterial, viral, and fungal infections.[11,107] An increased risk of pitted keratolysis, dermatophytosis, and verruca plantaris/vulgaris infections, as well as worsening of pompholyx[108] and eczematous dermatitis[10] have been reported.[11,88] In addition, HH contributes to body odor (bromhidrosis)[88,109-111] and sometimes poor posture in an effort to conceal perspiration.[112]

EFFECT ON QUALITY OF LIFE

Key points

- **Hyperhidrosis impairs daily functions and social interactions**
- **Depression and anxiety occur more commonly in patients with hyperhidrosis than in nonhyperhidrotic individuals**
- **The ability of patients to cope with hyperhidrosis does not seem to improve with time**

HH affects daily activities, work functions, and social interactions.[27] The impairment in quality of life related to HH is equivalent to that of severe psoriasis, rheumatoid arthritis, multiple sclerosis, and end-stage renal disease.[113,114] Limitations caused by primary HH include embarrassment, frustration, insecurity, low self-esteem, obstacles in establishing social and intimate relationships, and decreased leisure activities. Emotional state is moderately to severely affected in >50% of patients with HH.[101,115] Depression and anxiety occur more commonly in patients with HH than in healthy control subjects,[3] and 1 study reported that 63% of patients with HH felt unhappy or depressed.[16] It is often difficult to distinguish primary HH with secondary social anxiety from primary social anxiety disorder with secondary HH symptoms,[116] and some authors even recommend that HH treatment include antianxiety medications or cognitive behavioral therapy.[116-118] Moreover, 32% of patients with axillary HH described the condition to be hardly bearable or intolerable, which regularly or constantly negatively affected their daily activities.[3,27] Patients who suffer the most usually report HH for a longer duration of time,[115] indicating that the ability to cope with the problem does not improve with time.[27]

Resources for patients and health care professionals

Several resources provide information for patients with HH and health care professionals. The International Hyperhidrosis Society provides information through its website (SweatHelp.org),

e-newsletters, and seminars.[119] Hyperhidrosisuk.org is another resource that supplies information regarding the general nature of the condition and common treatment modalities.

Additional support websites, forums, and blogs for patients with HH include sweatyhands.proboards.com, socialphobiaworld.com/hyperhidrosis-forum, mylifeasapuddle.com, dailystrength.org/group/hyperhidrosis, www.checkyoursweat.com, hyperhidrosisnetwork.com, and more.

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