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PRIMARY HYPERHIDROSIS: EPIDEMIOLOGY, PATHOPHYSIOLOGY, CLINICAL FEATURES AND MODERN THERAPEUTIC APPROACHES — LITERATURE REVIEW

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Abstract: Primary hyperhidrosis (PHH) is a chronic disorder characterized by focal, excessive sweating beyond physiological thermoregulatory needs. Although not life-threatening, it significantly impairs quality of life, psychological well-being, and social functioning. This review summarizes current data on epidemiology, etiopathogenesis, clinical presentation, diagnostic criteria, and contemporary therapeutic strategies, including topical, systemic, and procedural interventions.

Keywords: Hyperhidrosis; Sweat Glands, Eccrine; Autonomic Nervous System Diseases; Sympathetic Nervous System; Botulinum Toxins, Type A; Cholinergic Antagonists; Iontophoresis; Thermotherapy; Thoracic Sympathectomy; Quality of Life.

Introduction

Hyperhidrosis is defined as excessive sweat production exceeding physiological requirements for thermoregulation. It is broadly classified into primary hyperhidrosis (PHH), which is idiopathic and focal in nature, and secondary hyperhidrosis (SHH), which results from systemic diseases, endocrine disorders, infections, malignancies, or pharmacological agents [1,2].

PHH is a chronic condition with onset typically in childhood or adolescence, and may persist throughout life. Despite its benign nature, it is associated with substantial psychosocial burden, including anxiety, social withdrawal, and reduced occupational productivity [3].

Epidemiology

The estimated prevalence of PHH varies between 1% and 5.3% globally, though underreporting is common due to stigma and lack of medical consultation [4]. Axillary hyperhidrosis is the most frequently reported subtype, followed by palmar, plantar, and craniofacial forms [5].

Studies indicate no strong gender predilection, although some reports suggest slightly higher healthcare-seeking behavior among females [6]. A positive family history is reported in up to 30–50% of cases, supporting a genetic predisposition with possible autosomal dominant inheritance with variable penetrance [7].

Pathophysiology and Etiopathogenesis

The exact pathophysiology of PHH remains incompletely understood. Current evidence suggests dysfunction of the sympathetic nervous system, particularly hyperactivity of postganglionic sympathetic cholinergic fibers innervating eccrine sweat glands [8].

Functional neuroimaging studies have demonstrated increased activity in hypothalamic regions responsible for thermoregulatory control, suggesting central dysregulation [9]. Genetic studies have identified potential susceptibility loci, including regions on chromosome 14q11.2–q13, although no single causative gene has been confirmed [10].

Eccrine glands in PHH patients are structurally normal, indicating that the disorder is functional rather than anatomical.

Clinical Manifestations

PHH is typically focal, bilateral, and symmetric. The most commonly affected regions include:

- Axillary region
- Palms
- Soles
- Craniofacial area

Symptoms are often triggered by emotional stress, heat, or spontaneous episodes without identifiable stimuli. Night sweating is generally absent, which helps differentiate PHH from secondary causes [11].

The condition significantly impacts daily life, including difficulties in writing, handshaking, use of electronic devices, and wearing certain clothing. Dermatological complications may include maceration, fungal infections, and irritant dermatitis [12].

Diagnostic Evaluation

Diagnosis is primarily clinical and based on established criteria proposed by the International Hyperhidrosis Society:

- Excessive focal sweating ≥6 months
- Bilateral and symmetrical distribution
- Impairment of daily activities
- Onset before 25 years
- Positive family history
- Cessation during sleep [13]

The **Hyperhidrosis Disease Severity Scale (HDSS)** is commonly used for grading severity and evaluating treatment response.

Objective tests include:

- Gravimetric measurement of sweat production

• Minor's iodine-starch test for localization

However, these are typically reserved for research or pre-procedural planning [14].

Treatment Strategies

1.1 Topical Therapy

First-line treatment includes topical aluminum chloride hexahydrate (10–20%), which obstructs eccrine sweat ducts. Its efficacy is well-established in mild-to-moderate axillary hyperhidrosis, although skin irritation is a common limiting factor [15].

Topical anticholinergics, such as glycopyrronium tosylate wipes, have shown efficacy in reducing axillary sweating with improved tolerability profiles [16].

1.2 Systemic Therapy

Systemic anticholinergic agents are indicated in moderate-to-severe PHH or multifocal disease.

• Oxybutynin

• Glycopyrrolate

Clinical studies demonstrate significant symptom reduction; however, adverse effects such as dry mouth, constipation, blurred vision, and urinary retention limit long-term use [17].

1.3 Iontophoresis

Tap water iontophoresis is particularly effective for palmar and plantar hyperhidrosis. It involves the transdermal delivery of ions using electrical current, reducing sweat gland activity. Maintenance therapy is typically required [18].

1.4 Botulinum Toxin Injections

Botulinum toxin type A inhibits acetylcholine release at the neuromuscular junction of eccrine glands. It is highly effective for axillary, palmar, and plantar hyperhidrosis, with effects lasting 4–9 months [19].

Limitations include pain during injection (especially palmar region) and the need for repeat treatments.

1.5 Energy-based and Device-assisted Therapies

Microwave thermolysis (e.g., targeted destruction of sweat glands) has demonstrated long-term efficacy in axillary hyperhidrosis [20]. Laser-based therapies and ultrasound-assisted techniques are emerging alternatives with promising results but limited long-term comparative data.

1.6 Surgical Treatment

Endoscopic thoracic sympathectomy (ETS) is reserved for severe, refractory cases. It involves interruption of sympathetic ganglia responsible for sweat production.

While ETS provides immediate and often permanent relief, complications include:

• Compensatory hyperhidrosis (most common)

• Gustatory sweating

- Neuralgia or neuropathic pain [21]

Due to these risks, careful patient selection is essential.

Impact on Quality of Life

PHH is strongly associated with reduced quality of life scores comparable to chronic dermatologic conditions such as psoriasis and eczema. Psychological comorbidities include social anxiety disorder, depressive symptoms, and reduced self-esteem [22].

Validated tools such as the Dermatology Life Quality Index (DLQI) are frequently used in clinical studies.

Conclusion

Primary hyperhidrosis is a multifactorial neurofunctional disorder with significant psychosocial burden. Despite its benign nature, it requires a structured diagnostic and therapeutic approach. Advances in pharmacological agents and minimally invasive procedures have significantly improved management outcomes, although long-term safety and comparative effectiveness remain areas for further research.

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