

Research Advances in Axillary Osmidrosis

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Abstract

Axillary osmidrosis (AO) is a chronic dermatological condition characterized primarily by persistent malodor in the axillary region. Its prevalence is relatively high among East Asian populations, and the condition can adversely affect patients' social functioning, self-esteem, and psychological well-being. In recent years, advances in molecular biology, microbiome research, and energy-based devices have improved understanding of the pathogenesis of AO and expanded available management strategies. This narrative review summarizes current evidence on the epidemiology and pathogenesis of AO, including genetic susceptibility, apocrine sweat gland function, cutaneous microbiota, and psychosocial aspects of odor perception. It also discusses diagnostic and assessment approaches and compares therapeutic strategies, including surgical procedures, energy-based interventions, botulinum toxin injections, topical agents, and emerging targeted therapies. Particular attention is paid to the strength and limitations of evidence supporting each modality and to treatment selection according to disease severity, concomitant hyperhidrosis, recurrence risk, and patient tolerance of scarring or repeated treatment. Future research directions are also discussed, with the aim of providing clinicians with a practical and evidence-informed reference for the diagnosis and management of AO.

Keywords

Axillary Osmidrosis, Axillary Bromhidrosis, ABCC11, Apocrine Sweat Gland, Cutaneous Microbiota, Therapeutic Advances

1. Introduction

Axillary osmidrosis (AO), also known as axillary bromhidrosis, is a chronic condition characterized by the production of unpleasant axillary odor. The malodor is mainly generated when secretions from axillary apocrine sweat glands are metabolized by resident skin microorganisms, resulting in volatile odoriferous com-

pounds on the skin surface. Although AO is not life-threatening, affected individuals may experience considerable psychosocial burden, including social avoidance, reduced self-esteem, embarrassment, and impaired quality of life. These effects may be particularly prominent in East Asian sociocultural contexts, where clinically perceptible axillary odor occurs in only a minority of the population. Previous research has shown that self-esteem in patients with AO is closely associated with perceived body odor, and that odor perception can influence how individuals are evaluated by others, highlighting the clinical and psychosocial importance of effective management [1].

The pathogenesis of AO involves genetic susceptibility, apocrine sweat gland activity, cutaneous microbiota, and host-microbe interactions. The identification of a single-nucleotide polymorphism (SNP) in the ATP-binding cassette subfamily C member 11 (ABCC11) gene has provided an important molecular basis for understanding the hereditary features of AO [2]. In parallel, therapeutic options have expanded from conventional radical surgery with large incisions to small-incision and minimally invasive procedures [3] [4], energy-based interventions such as laser, radiofrequency, and microwave systems [5] [6], botulinum toxin type A (BoNT-A) injections [7] [8], and topical or microbiota-directed formulations [9] [10]. However, the quality of evidence supporting these approaches varies substantially, and treatment selection remains influenced by disease severity, concomitant hyperhidrosis, recurrence risk, recovery time, and patient preference regarding scarring or repeated procedures.

This narrative review summarizes recent advances in the epidemiology, pathogenesis, diagnosis, assessment, and treatment of AO, drawing on relevant Chinese and international literature [6] [11] [12]. Particular attention is given to the strength and limitations of available evidence and to the practical selection of therapeutic strategies for different clinical scenarios.

2. Methods

This article is a narrative review rather than a formal systematic review. Relevant publications were identified through searches of PubMed, Web of Science, Google Scholar, and major Chinese academic databases, including CNKI and Wanfang, from database inception to [Month Year]. Search terms included “axillary osmidrosis,” “axillary bromhidrosis,” “ABCC11,” “apocrine sweat gland,” “cutaneous microbiota,” “botulinum toxin,” “microwave thermolysis,” “laser therapy,” “liposuction curettage,” “minimally invasive surgery,” and “topical therapy.” Articles were considered if they addressed the epidemiology, pathogenesis, diagnosis, assessment, or treatment of AO. Priority was given to clinical trials, comparative studies, long-term follow-up studies, reviews, and mechanistic studies directly relevant to axillary malodor. Publications focusing exclusively on generalized body odor, hyperhidrosis without osmidrosis, or non-axillary bromhidrosis were considered only when they provided relevant mechanistic or contextual information.

3. Epidemiology

The prevalence and social perception of AO vary markedly across ethnic groups. In many populations of European or African ancestry, functional ABCC11 protein is common, and axillary odor is often regarded as a normal physiological trait rather than a disorder requiring medical treatment. In contrast, the 538A allele of the ABCC11 gene, which results in the Arg180 variant, is highly prevalent in East Asian populations, including those in China, Japan, and South Korea. This allele promotes proteasomal degradation of the ABCC11 protein, impairs apocrine secretory function, and is associated with dry earwax and a very low risk of clinically perceptible axillary odor. Conversely, individuals carrying the wild-type 538G allele, which encodes the Gly180 variant and preserves ABCC11 function, are more likely to have wet earwax and an increased risk of AO [2].

In China, AO affects a substantial number of individuals and is a common reason for consultation in dermatology and plastic surgery clinics. Because clinically perceptible axillary odor represents a minority phenotype in many East Asian populations, affected individuals may experience disproportionate social pressure and a stronger demand for treatment. A retrospective review by Morioka *et al.* similarly suggested that AO constitutes a minority phenotype in the Japanese population, which may contribute to heightened psychosocial burden and treatment-seeking behavior [6]. Participants enrolled in clinical studies are commonly young or middle-aged adults, often between 18 and 45 years of age, and the condition affects both sexes. Some studies have reported a slightly higher proportion of female patients seeking medical care, which may reflect greater concern about abnormal body odor, different social expectations, or greater sensitivity to its interpersonal implications among women [12] [13].

4. Pathogenesis

4.1. Genetic Basis: ABCC11 Gene Polymorphism

The ABCC11 gene is located on chromosome 16q12.1 and encodes multidrug resistance-associated protein 8 (MRP8), a member of subfamily C of the ATP-binding cassette (ABC) transporter superfamily. The nonsynonymous single-nucleotide polymorphism c.538G>A (p.Gly180Arg) is currently regarded as the principal genetic determinant of AO. Biochemical studies have shown that the Arg180 variant accelerates proteasomal degradation of newly synthesized ABCC11 protein, resulting in reduced or absent functional transporter expression in apocrine sweat glands. Consequently, secretion of odor precursors, including dehydroepiandrosterone sulfate (DHEA-S) and sulfur-containing cysteine-glycine conjugates, is markedly reduced [2].

In contrast, wild-type ABCC11 carrying the Gly180 variant is functionally expressed in axillary apocrine sweat glands and facilitates the transport of multiple odor precursors into the glandular lumen. Using plasma membrane vesicles expressing ABCC11, Toyoda *et al.* established an *in vitro* transport assay and demonstrated that febuxostat, a drug approved for the treatment of gout, inhibited

ABCC11-mediated DHEA-S transport in a concentration-dependent manner, while exerting minimal effects on the homologous transporter ABCC2 [2]. These findings provide mechanistic proof of concept for ABCC11-targeted inhibition as a potential therapeutic strategy for AO. However, this evidence remains preclinical, and clinical efficacy, safety, optimal formulation, and delivery methods for AO have not yet been established.

Beyond ABCC11, structural and functional changes in sweat glands may also contribute to disease expression. Hatano *et al.* characterized histopathological alterations of apocrine sweat glands in patients with axillary osmidrosis and hyperhidrosis, providing morphological evidence linking sweat gland abnormalities with axillary malodor [14]. Nevertheless, the relative contribution of glandular morphology, secretory activity, and host-microbe interactions requires further clarification.

4.2. Apocrine Secretion and Odor Formation

The generation of axillary odor involves a multistep biochemical process. Apocrine sweat gland secretions are intrinsically odorless or only faintly odorous. After being released onto the skin surface, however, odorless precursor molecules are metabolized by cutaneous microorganisms, particularly *Corynebacterium* species, into volatile short-chain fatty acids (SCFAs) and volatile sulfur compounds (VSCs), which produce the characteristic malodor associated with AO [6] [10].

Major odor precursors include Cys-Gly-3M3SH [S-[1-(2-hydroxyethyl)]-L-cysteinyglycine conjugate], a thiol precursor that can be hydrolyzed by *Corynebacterium*-derived aminoacylase to release 3-methyl-3-mercapto-1-hexanol (3M3SH), which produces an onion-like or sulfurous odor. Fatty acid precursors secreted by apocrine sweat glands can also undergo hydrolysis by bacterial esterases to generate compounds such as 3-hydroxy-3-methylhexanoic acid (HMHA), which contributes to cheese-like or sweaty odor [6] [15]. Saito *et al.* reported that genistein, a soy isoflavone, inhibited enzymatic generation of these odoriferous molecules [16]. This finding supports the biological relevance of odor precursor metabolism, but it should be interpreted as mechanistic rather than clinical evidence for the treatment of AO.

4.3. Dysbiosis of the Cutaneous Microbiota

The cutaneous microbiota plays a pivotal role in the pathogenesis of AO. Under physiological conditions, the axillary microbial community is commonly dominated by *Staphylococcus* and *Corynebacterium*. Using high-throughput sequencing of the V4 region of 16S ribosomal DNA on the HiSeq 2500 platform, Zeng *et al.* compared the axillary microbiomes of patients with AO and healthy controls. They found that the relative abundance of *Corynebacterium* and anaerobic cocci was significantly higher in patients with AO, whereas the relative abundance of *Staphylococcus* was markedly lower [10]. These findings suggest that *Corynebac-*

terium and certain anaerobic bacteria may function as odor-associated taxa, whereas *Staphylococcus*, particularly *Staphylococcus epidermidis*, may have a protective or microbiota-stabilizing role.

The pH of the skin surface is an important determinant of microbial activity. In patients with AO, axillary skin pH has been reported to be elevated, with a mean value of 6.57 ± 0.72 , and is more alkaline than normal skin, which is generally below pH 5.0 [10]. This relatively alkaline microenvironment may favor the proliferation of odor-associated bacteria, including *Corynebacterium* species, thereby aggravating odor generation. Zeng *et al.* further identified a positive correlation between axillary pH and the relative abundance of *Corynebacterium*, whereas an inverse correlation was observed with the abundance of *Staphylococcus* [10]. These findings provide a rationale for modulating the axillary microenvironment, including skin pH, as a potential nonsurgical approach to AO.

Li *et al.* subsequently investigated the feasibility of treating AO through microbiota-rebalancing strategies and suggested that modulation of the cutaneous microbiota may represent a novel therapeutic strategy for nonsurgical management [9]. However, microbiota-directed therapy remains an emerging field. Larger controlled studies are needed to determine durability, optimal treatment protocols, recurrence rates, and whether changes in microbial composition translate into sustained clinical improvement.

Long-term use of antiperspirants may also alter the composition of the axillary microbiota. A retrospective study by Ho *et al.* suggested that prolonged antiperspirant use is associated with changes in axillary microbial diversity [17]. Aluminum salt-based antiperspirants temporarily suppress sweating by physically obstructing sweat gland ducts and may reduce odor indirectly by decreasing moisture and bacterial substrate availability. However, their effect is generally temporary, and odor may recur after discontinuation, particularly in patients with true AO rather than isolated hyperhidrosis.

4.4. Psychosocial Significance of Body-Odor Perception

Body odor functions as a chemosignal in human social interactions, and abnormal axillary odor may therefore have psychosocial consequences beyond local dermatological symptoms. Direct evidence in patients with AO suggests that perceived body odor is associated with self-esteem and interpersonal impressions [1]. Croijmans *et al.* found that perfume-use habits were positively associated with individual self-esteem and that body odor perception influenced first impressions, supporting the social relevance of odor perception and the potential psychosocial burden experienced by patients with AO [1].

Other studies on body odor and chemosignaling, although not conducted specifically in patients with AO, provide broader contextual support for the social significance of human odor perception. Schwambergova *et al.* reported that immune activation, such as that occurring after vaccination, could influence perceived body

odor and attractiveness ratings [18]. Schafer *et al.* investigated father-child body odor perception and suggested that body odor may contribute to recognition within familial relationships [19]. These findings highlight the biological and social roles of body odor but should be interpreted as indirect evidence rather than disease-specific data for AO.

5. Diagnosis and Assessment

5.1. Clinical Diagnostic Criteria

The diagnosis of AO is based primarily on clinical manifestations. The principal diagnostic feature is persistent abnormal axillary odor, particularly malodor that becomes more pronounced after physical activity, sweating, emotional stress, or prolonged occlusion of the axillary region. Associated features may include axillary hyperhidrosis, yellow staining of clothing in the axillary region, wet-type cerumen, and a positive family history.

Wet-type cerumen is a useful ancillary phenotypic marker because ABCC11 is also functionally expressed in the ceruminous glands of the external auditory canal. The presence of wet cerumen is strongly associated with functional ABCC11 genotype and may therefore support the clinical diagnosis of AO [2]. A positive family history is also relevant, as AO often shows an autosomal dominant inheritance pattern [6] [12]. Nevertheless, diagnosis should not rely on a single sign alone; odor severity, patient-reported symptoms, physical examination, family history, and possible concomitant hyperhidrosis should be considered together.

5.2. Assessment of Severity

Several methods are commonly used to assess the severity of AO and treatment response.

Lu Swab Method (LSM): After the axilla is fully exposed, the examiner rubs the axillary region 10 times with a cotton swab and evaluates the odor at a distance of 5-10 cm from the nose. Odor severity is graded on a four-point scale from 0 to 3: grade 0, no detectable odor; grade 1, mild malodor; grade 2, distinct malodor; and grade 3, pungent malodor. This method has been used in clinical studies by Zeng *et al.* and Li *et al.* because it is simple, noninvasive, and relatively reproducible [10] [13].

Visual Analog Scale (VAS): The VAS can be used to assess patient-perceived odor intensity, symptom-related distress, or satisfaction after treatment. Patients usually rate symptom severity on a scale from 0 to 10, with higher scores indicating greater odor intensity or distress. Because AO has a substantial subjective and psychosocial component, patient-reported outcomes are useful complements to examiner-based assessments [5] [11].

Hyperhidrosis Disease Severity Scale (HDSS): The HDSS is used primarily to evaluate the severity and daily-life impact of hyperhidrosis. It is not a specific severity scale for AO, but it may be useful when AO is accompanied by prominent axillary sweating. In such patients, HDSS scores can help determine whether sweat-

reducing therapies, such as antiperspirants, topical anticholinergics, or BoNT-A, are likely to provide additional benefit [8].

Genetic Testing: Detection of the ABCC11 c.538G>A variant using the SmartAmp method or polymerase chain reaction (PCR)-based sequencing may serve as an ancillary or confirmatory approach, particularly in patients with atypical clinical manifestations or uncertain diagnosis [2]. However, genetic testing should be interpreted in conjunction with clinical findings, because the presence of a functional ABCC11 genotype indicates susceptibility rather than the absolute severity of malodor.

5.3. Differential Diagnosis

AO should be differentiated from other causes of axillary or generalized malodor. Axillary hyperhidrosis is characterized primarily by excessive sweating rather than distinctive malodor. Although sweating may aggravate odor by increasing moisture and bacterial metabolism, hyperhidrosis and AO are not equivalent conditions, and they may occur independently or coexist [8] [14]. Secondary malodor associated with contact dermatitis, eczema, intertrigo, or bacterial/fungal infection should also be considered, especially when erythema, scaling, maceration, pain, or discharge is present.

Axillary chromhidrosis is another differential diagnosis and is characterized by visibly colored sweat caused by pigment secretion from apocrine glands or by exogenous chromogenic substances [6] [15]. Fish odor syndrome, or trimethylaminuria, is a systemic metabolic disorder that causes generalized fish-like body odor due to impaired trimethylamine metabolism, rather than malodor limited mainly to the axillary region [15]. Careful history taking, physical examination, odor distribution, associated skin findings, and, when necessary, laboratory testing can help distinguish these conditions.

6. Treatment

Therapeutic strategies for AO can be broadly classified into conservative or non-surgical interventions, energy-based device therapies, BoNT-A injections, and surgical procedures. Available treatment modalities have been reviewed by Malik *et al.* [12] [15], and several clinical studies have compared the long-term efficacy and safety of different approaches [6] [11] [13]. However, the level of evidence varies substantially among treatment categories. Surgical procedures and energy-based devices are supported mainly by retrospective studies, prospective case series, and a limited number of comparative studies. Topical acidified fatty acid esters have been evaluated in a small randomized controlled trial, whereas microbiota-rebalancing strategies remain at an early stage of clinical investigation. BoNT-A has strong evidence for primary axillary hyperhidrosis, but evidence supporting its use for isolated AO is less robust and largely indirect. ABCC11-targeted inhibitors and genistein remain mechanistic or preclinical strategies, and their clinical efficacy in AO has not yet been established. Therefore, treatment selection should

be individualized according to odor severity, concomitant hyperhidrosis, recurrence risk, recovery time, patient expectations, and tolerance of scarring or repeated treatment.

6.1. Conservative Treatment

6.1.1. Topical Antiperspirant and Deodorant Preparations

Topical antiperspirants, antimicrobial agents, deodorants, and fragrances are commonly used to reduce axillary sweating, suppress odor-associated bacteria, neutralize malodorous compounds, or mask unpleasant odor. In general, these topical approaches are most suitable for patients with mild AO, patients with concomitant axillary hyperhidrosis, or individuals who are unwilling to undergo procedural treatment. Their efficacy is usually limited in moderate-to-severe AO because they do not reliably eliminate apocrine sweat glands or permanently suppress odor precursor secretion. In addition, therapeutic effects are typically temporary, and prolonged use may cause local adverse effects such as irritation, contact dermatitis, hyperpigmentation, or hyperkeratosis [12].

Topical anticholinergic agents inhibit acetylcholine-mediated stimulation of eccrine sweat glands and are approved or studied primarily for primary axillary hyperhidrosis rather than AO itself. For example, topical glycopyrronium formulations have been approved by the US Food and Drug Administration for primary axillary hyperhidrosis in patients aged 9 years or older. Their potential benefit in patients with AO is likely indirect and may be most relevant when malodor is accompanied by excessive sweating. At present, direct clinical evidence supporting topical anticholinergics as a specific treatment for isolated AO remains limited. Reported adverse effects may include dry mouth, blurred vision, mydriasis, urinary retention, and local skin irritation [20].

Aluminum salt-based antiperspirants, typically formulated as 20%-25% aluminum chloride in ethanol, reduce perspiration by physically obstructing the ductal openings of eccrine sweat glands. By decreasing axillary moisture, they may indirectly reduce bacterial activity and the availability of substrates for odor generation. However, their direct effect on apocrine sweat glands is limited, and sustained application is required because the benefit is short-lived and recurrence is common after discontinuation [15]. Ho *et al.* reported that prolonged antiperspirant use was associated with alterations in axillary microbial composition, suggesting that their mechanism may extend beyond ductal obstruction to include modulation of the local cutaneous microbial ecosystem [17]. Nevertheless, these products should be considered supportive or adjunctive measures rather than definitive treatment for moderate-to-severe AO.

6.1.2. Topical Acidified Fatty Acid Esters

In a prospective randomized controlled trial, Zeng *et al.* randomly assigned 32 patients with AO to receive acidified fatty acid esters or conventional fatty acid esters as the control intervention [10]. Preliminary findings showed that both the cure rate and overall response rate at 4 weeks were significantly higher in the acid-

ified fatty acid ester group than in the control group. During the subsequent 16-week treatment period, the cure rate increased significantly between weeks 4 and 16 and remained relatively high at the 3-month follow-up visit. Mechanistic analyses suggested that acidified fatty acid esters reduced axillary skin pH from 6.57 ± 0.72 to 6.15 ± 0.51 ($P < 0.001$) and reshaped the cutaneous microbial ecosystem [10].

These findings suggest that noninvasive modulation of the axillary microenvironment may alleviate AO, particularly in patients with mild-to-moderate disease who are unwilling to undergo surgery. Li *et al.* further supported the feasibility of microbiota-directed treatment from the perspective of restoring cutaneous microbial homeostasis [9]. However, the available clinical evidence remains limited by small sample size and relatively short follow-up. Larger randomized controlled trials are needed to determine durability, recurrence rates, optimal treatment duration, maintenance protocols, and comparative efficacy against established treatments.

6.1.3. Soy Isoflavone Genistein

Saito *et al.* reported that the soy isoflavone genistein inhibited the generation of odoriferous molecules associated with AO [16]. As a phytoestrogen, genistein may interfere with enzymatic metabolism of odor precursors and may also influence the secretory activity of apocrine sweat glands. These findings provide mechanistic support for a potential natural-product-based strategy for reducing axillary malodor. However, the current evidence should be interpreted as preclinical or mechanistic rather than clinical. Well-designed human studies are required before genistein can be recommended as a standard treatment for AO [16].

6.1.4. ABCC11-Targeted Inhibitors

As discussed above, Toyoda *et al.* showed *in vitro* that febuxostat inhibited ABCC11-mediated transport activity, with a half-maximal inhibitory concentration (IC_{50}) of $3.26 \mu\text{mol/L}$ [2]. This result raises the possibility that febuxostat, an already approved drug, could be repurposed as an ABCC11-targeted treatment for AO. However, the available evidence is limited to an *in vitro* study, and its therapeutic efficacy in patients with AO has not yet been evaluated. Further research is therefore needed to develop suitable topical formulations, determine whether adequate drug concentrations can be achieved within the target skin tissue, and assess local and systemic safety. Dose optimization and early proof-of-concept clinical trials in patients with clinically significant AO will also be necessary before this approach can be considered for routine clinical use.

6.2. Botulinum Toxin Type A Injection

6.2.1. Mechanism of Action and Indications

BoNT-A inhibits acetylcholine release from cholinergic nerve terminals, thereby suppressing cholinergic stimulation of sweat glands. It has well-established efficacy in primary axillary hyperhidrosis. In patients with AO, BoNT-A may reduce

malodor indirectly by decreasing sweating, axillary moisture, and the amount of substrate available for bacterial metabolism. Its benefit is therefore expected to be greater in patients with AO accompanied by prominent axillary hyperhidrosis than in those with isolated malodor.

BoNT-A injection may be considered for patients with mild-to-moderate AO with concomitant hyperhidrosis, patients who are unwilling or unsuitable to undergo surgery, and selected patients with residual or recurrent symptoms after surgical or energy-based treatment [7] [8]. However, clinicians should explain that BoNT-A is not a definitive gland-removing procedure and that repeated injections are usually required.

6.2.2. Injection Protocol

According to expert consensus recommendations on BoNT-A injection therapy for hyperhidrosis and AO, the injection area usually corresponds to the region affected by excessive sweating or malodor. The sweating field may be delineated using the iodine-starch test (Minor test), particularly when hyperhidrosis is prominent. A commonly used protocol involves a total dose of 50-100 U per axilla for onabotulinumtoxinA or 100-200 U per axilla for Hengli, a domestically manufactured Chinese formulation, administered through evenly distributed multipoint injections at intervals of 1-2 cm, with 2-5 U delivered at each injection site [7] [8]. The injection depth generally ranges from the deep dermis to the superficial subcutaneous layer, while excessively deep injection into the muscular layer should be avoided. Therapeutic effects usually persist for 4-12 months, and periodic repeat injections may be required [7] [8].

6.2.3. Efficacy and Safety

BoNT-A injection has a strong evidence base for axillary hyperhidrosis, with reported response rates exceeding 80% in many studies. In contrast, its effectiveness for isolated AO without concomitant hyperhidrosis is less certain. This limitation may be related to the complex regulation of apocrine sweat glands, which involves not only cholinergic pathways but also adrenergic influences, hormonal factors, and microbial metabolism [8]. Therefore, BoNT-A should be regarded primarily as a sweat-reducing and symptom-relieving treatment rather than a curative therapy for AO. Common adverse events include injection-site pain, transient ecchymosis, local discomfort, temporary muscle weakness, and compensatory sweating; serious complications are rare [7] [8].

6.3. Energy-Based Device Therapy

6.3.1. Nd:YAG Laser

Geng *et al.* reported a clinical study of percutaneous interstitial neodymium-doped yttrium aluminum garnet (Nd:YAG) laser therapy at a wavelength of 1,064 nm for AO [5]. In this procedure, a fine laser fiber is introduced percutaneously to a depth of approximately 2-3 mm below the skin. Under ultrasound guidance or after tumescent anesthesia, apocrine sweat glands are thermally damaged through

laser-induced photothermal effects, while residual glandular tissue may be coagulated [5]. Compared with conventional surgery, percutaneous interstitial laser therapy has potential advantages, including reduced tissue trauma, faster recovery, and less visible scarring.

The study reported an overall response rate exceeding 85% after a single treatment session, with a low recurrence rate and high patient satisfaction [5]. Reported complications included transient subcutaneous induration, mild thermal injury, and temporary hypoesthesia, most of which resolved spontaneously. In a long-term follow-up study, Chen *et al.* compared subcutaneous laser therapy with superficial liposuction curettage and found comparable sustained efficacy, while the laser group had a shorter recovery period and fewer procedure-related complications [11]. Nevertheless, evidence for laser therapy is still based mainly on clinical series and comparative observational studies, and further prospective controlled studies are needed to define optimal energy parameters, patient selection, and long-term recurrence rates.

6.3.2. Microwave Thermolysis

Microwave thermolysis, delivered using the miraDry system, emits microwave energy at a frequency of 5.8 GHz to generate a confined thermal effect at the dermal-subcutaneous tissue interface, approximately 2-5 mm below the skin surface. This energy can irreversibly damage apocrine and eccrine sweat glands, while an integrated cooling system protects the epidermis [6]. Because both odor generation and sweating may be reduced, microwave therapy may be particularly useful in patients with AO accompanied by axillary hyperhidrosis.

Morioka *et al.* reported response rates of approximately 70%-90% for microwave thermolysis in the treatment of AO [6]. Many patients require two treatment sessions to achieve optimal outcomes, and the effect may be durable because treated sweat glands are irreversibly destroyed. Common adverse events include edema in the treated area, localized pain, bruising, numbness, and transient sensory disturbance. Reduced axillary hair growth may also occur in some patients. Serious complications, such as thermal skin injury and nerve damage, are uncommon when the procedure is performed appropriately [6]. Malik *et al.* also recognized microwave thermolysis as an important nonsurgical option within the multimodal treatment framework for AO [15]. However, high-quality comparative studies with long-term follow-up remain limited.

6.4. Surgical Treatment

Surgical treatment aims to physically remove or destroy apocrine gland-bearing tissue in the axilla and remains the most definitive therapeutic approach for moderate-to-severe AO. It is primarily indicated for patients with severe malodor, inadequate response to conservative therapy, frequent recurrence after nonsurgical treatment, or a strong preference for a potentially durable intervention [12]. Surgical outcomes depend on the completeness of gland removal, preservation of skin flap viability, compression dressing, postoperative immobilization, and surgeon

experience. Based on incision size and operative technique, the principal surgical procedures can be classified as follows.

6.4.1. Conventional Radical Surgery through a Large Incision

Conventional radical surgery involves direct excision of subcutaneous adipose tissue and dermal tissue containing abundant apocrine sweat glands through a fusiform or S-shaped axillary incision, usually measuring 3-8 cm. Gland-bearing skin may also be excised when necessary to achieve more complete removal. Although this procedure can provide a high definitive response rate, it is associated with relatively long incisions, conspicuous scarring, prolonged recovery, and a greater risk of postoperative complications, including skin flap necrosis, infection, hematoma, and contracture [6] [12]. Because of these disadvantages, conventional radical surgery has been progressively replaced by minimally invasive techniques and is now generally reserved for extremely severe disease or recurrent cases in which other surgical modalities have failed.

6.4.2. Subcutaneous Trimming through a Small Incision

Subcutaneous trimming through a small incision is one of the most widely used minimally invasive surgical procedures for AO. The technique involves creating one or two small incisions, usually 0.5-1.5 cm long, within the axillary crease. After the skin is separated from the underlying subcutaneous tissue, sweat gland-bearing tissue along the deep dermal surface and superficial fascial layer is removed using tissue scissors or a curette, either under limited visualization or with direct visual guidance. The skin flap is then secured with an appropriate compression dressing [3]. Compared with conventional large-incision surgery, this approach offers a more concealed incision, less conspicuous scarring, and a shorter recovery period, usually approximately 7-14 days, resulting in improved patient satisfaction.

Xiong *et al.* modified the conventional small-incision curettage technique by adopting a dual-incision design comprising an incision along the inferior axillary margin and a second incision at the lateral border [21]. The lateral incision facilitates operative manipulation and reduces the need for the surgeon to change position repeatedly. In an invited response, Yang *et al.* noted that although the additional incision improved procedural convenience, its location outside the axillary crease could result in a more conspicuous scar [22]. They also emphasized that a short follow-up period of 30 days was insufficient to evaluate scar formation and that prevention of postoperative hematoma requires comprehensive consideration of multiple factors, including sex, coagulation status, compression dressing, and restriction of upper-limb movement [22].

6.4.3. Small-Incision Subcutaneous Curettage Combined with Suction

Small-incision subcutaneous curettage combined with suction integrates mechanical curettage and negative-pressure aspiration. After glandular tissue is disrupted by curettage, it is removed by suction, which may allow more complete clearance of subcutaneous sweat glands and reduce the likelihood of residual tissue being

overlooked during blind dissection [4]. Reported response rates are approximately 90%, and the incidence of complications appears comparable to that associated with small-incision subcutaneous trimming alone [4]. However, available data are derived mainly from clinical series, and outcomes may vary according to the extent of curettage, operator experience, and postoperative care.

6.4.4. Tumescant Liposuction Combined with Power-Assisted Curettage

Lin *et al.* reported experience with tumescent anesthesia combined with power-assisted liposuction curettage for the treatment of AO [23]. Infiltration of tumescent solution, a large volume of dilute solution containing epinephrine and lidocaine, expands the subcutaneous tissue, reduces intraoperative bleeding, and facilitates separation of glandular tissue from surrounding structures. A power-assisted device equipped with a vibrating cannula may improve the uniformity and completeness of sweat gland removal. The study reported favorable response rates and relatively low incidences of complications such as hematoma and skin flap necrosis compared with conventional techniques [23]. These findings support this method as a refinement of minimally invasive surgery, although further comparative studies are needed to confirm its long-term superiority over other small-incision procedures.

6.4.5. Hidden-Blade Scalpel-Assisted Technique

Lu *et al.* reported the use of a hidden-blade scalpel as an auxiliary instrument in surgery for AO [24]. In this specially designed device, the blade remains concealed within a protective sheath and is deployed after the instrument is introduced into the subcutaneous plane through a small incision. This design permits controlled dissection of subcutaneous glandular tissue while helping maintain a stable cutting depth. Compared with conventional tissue scissors, the hidden-blade scalpel may facilitate manipulation in anatomically difficult corner regions, particularly along the axillary margins, thereby reducing residual sweat glands and lowering recurrence risk [24]. The technique is promising, but its advantages should be confirmed in larger comparative studies with longer follow-up.

6.4.6. Versajet-Assisted Hydrosurgical Gland Removal

Ho reported a small-incision technique for treating AO using the Versajet hydro-surgery system for hydraulic epilation [25]. By generating a high-velocity water jet based on the Venturi effect, the Versajet system enables precise dissection and simultaneous aspiration of soft tissue. This approach may allow selective removal of subcutaneous glandular tissue and hair follicles under assisted visualization while minimizing injury to surrounding structures. Because hair follicles and apocrine glands are anatomically associated, follicular removal may concomitantly reduce residual apocrine glands. Preliminary findings indicate a relatively high response rate and an acceptable complication profile, although larger studies with long-term follow-up are needed to evaluate recurrence, scarring, sensory disturbance, and patient satisfaction [25].

6.4.7. Comparative Evaluation of Modified Minimally Invasive Surgical Techniques

In a retrospective study, Li *et al.* compared the long-term outcomes of multiple surgical and nonsurgical modalities [13]. Surgical treatment was generally more effective than nonsurgical intervention, although differences in efficacy among various operative techniques were limited. These findings suggest that, beyond the choice of technique itself, surgeon experience, adequate removal of gland-bearing tissue, appropriate patient selection, and postoperative management are important determinants of clinical outcomes [13]. At present, small-incision procedures provide a balance between efficacy and cosmetic acceptability and are widely used for patients with moderate-to-severe AO.

6.5. Comparative Evaluation and Selection of Treatment Modalities

Malik *et al.* summarized available therapeutic modalities for AO in a comprehensive literature review [15]. In a long-term follow-up study exceeding 12 months, Chen *et al.* found no significant difference in sustained efficacy between subcutaneous laser therapy and superficial liposuction curettage, although the two approaches may be suited to different patient populations [11]. Overall, surgical treatment, particularly minimally invasive procedures performed through small incisions, remains the most consistently effective and durable definitive treatment currently available [3] [4] [21] [23]. Energy-based interventions, including laser and microwave therapies, are suitable for patients who prefer less invasive treatment, shorter recovery, or less visible scarring, although recurrence and the possibility of repeated treatment should be discussed [5] [6] [11]. BoNT-A injection is most appropriate for mild-to-moderate AO accompanied by prominent hyperhidrosis, whereas its effect on isolated malodor may be limited [7] [8]. Topical preparations, including antiperspirants, deodorants, acidified fatty acid esters, and microbiota-directed approaches, may be considered for mild disease, maintenance therapy, or adjunctive management of postoperative recurrence [9] [10].

In clinical practice, treatment selection for AO should be tailored to disease severity, the presence of concomitant hyperhidrosis, recurrence risk, expected recovery time, and the patient's willingness to accept scarring or repeated treatment. For mild AO, particularly in patients who are reluctant to undergo procedural intervention, topical deodorants, antiperspirants, acidified fatty acid esters, and other microbiota-directed therapies may be considered. Their effects, however, are often temporary, and repeated application or maintenance treatment is usually necessary.

When prominent hyperhidrosis is also present, antiperspirants, topical anticholinergics, and BoNT-A may offer additional benefit by reducing sweat production, although their effect on malodor alone may be less pronounced. Energy-based modalities, such as laser therapy and microwave thermolysis, may be suitable for patients who prefer a minimally invasive approach, less visible scarring, and a shorter recovery period. These patients should nevertheless be informed

that recurrence may occur and that additional treatment sessions may be required.

For moderate-to-severe AO, as well as recurrent disease that has not responded adequately to conservative treatment, small-incision surgical removal of apocrine gland-bearing tissue remains the most durable option. It is particularly appropriate for patients who prioritize long-term efficacy and are willing to accept potential complications, including scarring, hematoma, sensory disturbance, and temporary limitation of upper-limb movement.

7. Quality of Life and Psychosocial Impact Associated with Axillary Osmidrosis

The impairment of quality of life (QoL) among patients with AO warrants clinical attention. Because axillary odor is socially noticeable and often difficult for patients to conceal, affected individuals may experience embarrassment, social anxiety, avoidance of close interpersonal contact, reduced self-esteem, and depressive symptoms [1] [6]. These effects may be amplified in sociocultural contexts where clinically perceptible axillary odor is uncommon or stigmatized.

Current evidence suggests that the psychosocial burden of AO is related not only to objective odor intensity but also to patients' subjective perception of odor, concern about being evaluated by others, and previous negative social experiences [1]. Direct evidence from studies involving patients with AO supports an association between perceived body odor, self-esteem, and interpersonal impressions [1]. Broader studies on human body-odor perception and chemosignaling further suggest that body odor can influence social evaluation and interpersonal recognition, although such findings should be regarded as indirect contextual evidence rather than disease-specific data for AO [18] [19].

Therefore, clinical management of AO should extend beyond biological odor reduction. Clinicians should assess symptom severity, patient-reported distress, social avoidance, and expectations for treatment. For patients with marked anxiety, depressive symptoms, excessive preoccupation with odor, or impairment in work, study, or interpersonal relationships, psychological counseling or referral for mental health support may be appropriate. Integrating effective odor control with psychosocial assessment and support may improve overall treatment satisfaction and QoL.

8. Future Perspectives

Based on the current state of research on AO, future investigations should focus on the following areas.

Future research on AO should move beyond descriptive findings and place greater emphasis on translational and clinically applicable strategies. One important direction is the development of ABCC11-targeted therapies. The *in vitro* study by Toyoda *et al.* has provided an important mechanistic basis for pharmacological inhibition of ABCC11 [2]. Nevertheless, several issues still need to be resolved before this strategy can be considered for clinical use. These include the design of

suitable topical delivery systems, improvement of cutaneous penetration, maintenance of sufficient local drug concentrations, and careful evaluation of both local and systemic safety. Well-designed proof-of-concept clinical trials will also be necessary. In this context, topical ABCC11-targeted formulations, such as febuxostat-containing preparations, nanoemulsions, and microneedle-assisted delivery systems, may offer useful directions for further investigation, although their clinical efficacy has not yet been confirmed.

Another area that deserves closer attention is precision modulation of the cutaneous microbiota. With the continued development of metagenomic and metabolomic techniques, it should become possible to characterize the axillary microbiome and odor-related metabolites at a much higher resolution. Such work may help identify key odor-associated bacterial taxa and metabolic pathways involved in AO [9] [10]. These findings could, in turn, support the development of microbiota-directed interventions, including targeted antimicrobial treatment, probiotic approaches, bacteriophage-based therapies, and pH-modulating formulations. However, future studies should not only describe microbial changes after treatment, but also clarify whether these changes are causally linked to clinical improvement and whether the effects can persist after treatment is discontinued. There is also a clear need for long-term randomized controlled trials and high-quality comparative studies. At present, many therapeutic studies are constrained by small sample sizes, single-center designs, short follow-up periods, inconsistent outcome measures, and a limited number of high-quality RCTs [11] [13] [15]. In particular, head-to-head comparisons between energy-based devices and minimally invasive surgical procedures would be valuable for guiding clinical decision-making. Multicenter studies are also needed to evaluate the long-term efficacy, safety, recurrence rates, and maintenance strategies of topical treatments and microbiota-directed therapies.

In addition, future research should work toward more standardized and comprehensive assessment frameworks. Current methods for evaluating treatment outcomes in AO remain relatively inconsistent. Subjective odor-rating tools, such as the Lu Swab Method (LSM) and the Visual Analog Scale (VAS), can be affected by evaluator experience, environmental conditions, patient perception, and cultural background [10] [13]. Objective approaches, including electronic-nose technology, gas chromatography-mass spectrometry (GC-MS), and standardized volatile-compound profiling, may help improve the comparability of findings across studies. Ideally, future assessment systems should integrate objective odor measurement, clinician-rated severity, patient-reported outcomes, recurrence evaluation, adverse-event reporting, and quality-of-life assessment.

Finally, psychosocial care should be more fully incorporated into both clinical management and future research. AO can impose a considerable psychosocial burden, as suggested by direct evidence from patients with AO and indirect evidence from broader studies of human body-odor perception [1] [18] [19]. Therefore, future efficacy evaluations should include validated psychological and qual-

ity-of-life instruments, such as the Dermatology Life Quality Index (DLQI), anxiety and depression screening tools, and treatment satisfaction scales. For patients who experience marked social avoidance, anxiety, depressive symptoms, or excessive concern about body odor, integrated dermatology-psychology models of care may be particularly useful.

9. Conclusions

Axillary osmidrosis is a chronic dermatological condition with a multifactorial pathogenesis. Its development involves genetic susceptibility, particularly ABCC11 gene polymorphisms [2], apocrine sweat gland activity [14], the cutaneous microbiota and metabolism of odor precursors [9] [10], as well as the psychosocial burden associated with perceived axillary odor. The psychosocial impact of AO is supported by studies conducted directly in patients with the condition, together with broader evidence concerning the perception of human body odor [1] [18] [19].

For moderate-to-severe AO, surgical treatment remains the most consistently effective and durable option, particularly when apocrine gland-bearing tissue is removed through a small incision using minimally invasive techniques [3] [4] [21] [23]. Energy-based modalities, such as neodymium-doped yttrium aluminum garnet (Nd) laser therapy and microwave thermolysis, offer less invasive alternatives for patients who place greater importance on shorter recovery and less visible scarring. However, the possibility of recurrence and the potential need for additional treatment sessions should be discussed beforehand [5] [6] [11]. BoNT-A injection may be more suitable for patients with mild-to-moderate AO accompanied by prominent hyperhidrosis, whereas its effect on malodor in the absence of excessive sweating remains less clearly established [7] [8]. Topical microbiota-modulating preparations, including acidified fatty acid esters, represent another noninvasive option, although larger studies with longer follow-up are needed to clarify the durability of their effects and appropriate maintenance regimens [10]. ABCC11-targeted inhibitors may offer a more mechanism-based approach, but the available evidence remains preclinical, and their clinical efficacy in AO has not yet been demonstrated [2].

In clinical practice, treatment plans should be tailored to odor severity, concomitant hyperhidrosis, recurrence risk, psychological distress, expected recovery, and the patient's willingness to accept scarring or repeated treatment. A multimodal strategy may be appropriate in selected cases. Further progress will depend on higher-quality comparative studies, more consistent outcome assessment, objective methods of odor measurement, continued development of microbiome-based interventions, and closer integration of psychosocial care into the management of AO.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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