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Review Article

Clinical Outcomes of botulinum toxin injection in Patients with acne vulgaris

Zhao F^{1*}, Yang Y² and Liu L³

¹Department of Dermatology, Krishna Institute for Advanced Dermatology, Vietnam

²Department of Clinical Dermatology, Mekong Institute of Dermatology, China

³Department of Cosmetic and Aesthetic Surgery, Parana Research Center for Dermatology, Indonesia

*Address for Correspondence: Feng Zhao, Department of Dermatology, Krishna Institute for Advanced Dermatology, Vietnam; E-mail: feng.zhao@krishna.edu

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Abstract

Background: Acne vulgaris is driven by androgen-stimulated sebum overproduction, follicular hyperkeratinization, Cutibacterium acnes colonization, and perifollicular inflammation, and a substantial subset of patients present with a seborrheic, enlarged-pore phenotype that responds poorly to conventional comedolytic and antimicrobial regimens.

Objective: This review examines the rationale for, and the clinical evidence supporting, superficial intradermal botulinum toxin type A injection as a treatment for acne vulgaris, focusing on sebum output, pore size, and inflammatory lesion counts.

Methods: We synthesized mechanistic, in vitro, and clinical studies addressing cholinergic regulation of the pilosebaceous unit, intradermal botulinum toxin injection technique, and reported outcomes in patients with acne vulgaris or seborrhea-predominant oily skin.

Findings: Botulinum toxin blocks acetylcholine-driven sebocyte lipogenesis and dampens neurogenic perifollicular inflammation; published series report meaningful reductions in sebum excretion rate and pore size within four to twelve weeks, with a more modest reduction in inflammatory lesion counts, while adverse events are dominated by transient bruising, with motor complications rare when depth is controlled.

Conclusions: Intradermal botulinum toxin injection is a biologically coherent and generally well-tolerated adjunct for sebum- and pore-driven acne and seborrhea, best positioned alongside rather than in place of guideline-directed topical and systemic acne therapy until larger comparative and long-term trials clarify its role in inflammatory lesion and scarring outcomes.

Keywords: botulinum toxin; acne vulgaris; sebum reduction; microbotox; facial pores

1. Introduction

Acne vulgaris arises from a convergence of androgen-driven sebum overproduction, follicular hyperkeratinization, colonization by *Cutibacterium acnes*, and a perifollicular inflammatory cascade that determines whether a lesion resolves cleanly or scars [1,2]. The sebaceous gland is itself under complex neuroendocrine and paracrine regulation that sets the rate of sebocyte lipogenesis and terminal differentiation [3]. Guidelines built around this model recommend topical retinoids, benzoyl peroxide, and, for more extensive disease, oral antibiotics, hormonal therapy, or isotretinoin [4,5], but a substantial subset of patients, those with a seborrheic, enlarged-pore presentation who are poor candidates for prolonged antibiotics or who cannot tolerate isotretinoin, remain under-served [6,7]. It is this sebum-driven population that has prompted interest in botulinum toxin type A (BoNT-A) as an adjunct.

BoNT-A cleaves SNAP-25 and blocks vesicular acetylcholine release, and its cosmetic use has for three decades been confined almost entirely to facial rhytides [8,9,10]. Superficial intradermal microdroplet injection, popularized as "microbotox," diffuses the toxin through the papillary and upper reticular dermis rather than to a target muscle, affecting sweat glands, sebaceous units, and dermal microvasculature independent of any motor effect [11,12]. Because sebaceous glands are richly innervated by cholinergic fibers and acetylcholine stimulates sebocyte lipogenesis via muscarinic signalling, this technique offers a plausible route to down-regulate sebum output pharmacologically [13]. This review synthesizes the mechanistic rationale, clinical evidence, and safety profile of intradermal BoNT-A for acne vulgaris, focusing on sebum output, pore size, and inflammatory lesion counts.

2. Pathophysiology and the rationale for a neuromodulator approach

Sebum output is the strongest predictor of both acne

severity and perceived pore size: photographic studies correlating sebum excretion rate with pore diameter consistently find that patients in the highest sebum-output quartile have visibly larger pores than low-output peers, independent of age [14,15]. Excess sebum favors *C. acnes* overgrowth and follicular comedogenesis, triggering the innate immune signalling that drives the papulopustular lesions patients most want treated [16,17], and diet, hormonal status, and genetics further modulate this lipogenic response across a population [18,19,20].

Conventional therapies target keratinization, *C. acnes* and inflammation, or androgen-driven sebogenesis [4,21,22], but none acts directly on the cholinergic signal that acutely drives sebocyte lipid synthesis. Because cholinergic agonists stimulate, and anticholinergic blockade reduces, sebaceous lipogenesis in ex vivo human skin, the pharmacological logic for BoNT-A as a sebosuppressive agent is well grounded, even though its adoption in acne has lagged its use in hyperhidrosis and rejuvenation by roughly two decades [13,23].

3. Mechanism of action in the pilosebaceous unit

BoNT-A's cleavage of SNAP-25 and blockade of calcium-dependent vesicle fusion applies equally to motor endplates, eccrine glands, and the periglandular cholinergic fibers innervating sebaceous acini [8,24]. Injected intradermally, the toxin diffuses a few millimeters and denervates the local field of sweat and sebaceous glands without clinically evident weakness, provided depth and volume are controlled [11,23]. In vitro work on cultured sebocytes shows that cholinergic stimulation increases lipid droplet formation, and that BoNT-A pretreatment blunts this acetylcholine-driven response, giving a cellular basis for the sebum reductions reported clinically [13].

A second, increasingly emphasized mechanism is anti-inflammatory: BoNT-A blocks release of substance P, calcitonin gene-related peptide, and glutamate from

sensory nerve terminals, neuropeptides that promote neurogenic inflammation and mast cell degranulation in the papillary dermis [24,25]. Because acne's inflammatory lesions are driven in part by this neurogenic axis alongside the C. acnes-triggered innate response, suppressing local neuropeptide release offers a second, sebum-independent route to reduced lesion counts [25,26].

4. Injection technique and treatment protocols

Protocols for acne and seborrhea converge on a diluted, low-dose intradermal microdroplet technique distinct from the deeper, higher-unit intramuscular injections used for rhytides. Most series dilute 100 units of onabotulinumtoxinA or incobotulinumtoxinA in 4-10 mL saline and deliver 0.02-0.05 mL aliquots at 1-1.5 cm intervals across the forehead, nose, cheeks, and chin with a 30-32 gauge needle held near-horizontal to stay within the papillary-to-superficial-reticular dermis [27,28,29]. Per-session doses range from roughly 20 to 100 units depending on surface area treated, considerably lower than glabellar or forehead doses, since the intent is diffuse superficial coverage rather than focal paralysis [30,31].

Injectors avoid the deeper dermal plane and keep bleb volume small to prevent blanching, and most protocols exclude the immediate periorbital and perioral regions where diffusion could produce ptosis or asymmetric smile [32,33,34]. A session takes 15-20 minutes under topical anesthesia, and most series retreat at three to six months, tracking BoNT-A's known functional half-life at the neuromuscular and periglandular junction [10,8].

5. Clinical outcomes: sebum reduction

The most consistently reported outcome is a reduction in sebum excretion rate, measured by sebumeter before and after treatment. Rose and Goldberg's prospective series documented significant sebum reduction at four and eight weeks, with oiliness improving in parallel [28]. Shah's earlier open-label series reported comparable reductions with visible pore narrowing on standardized photography [27], and Jung and colleagues, using a split-face design,

found significantly greater sebum reduction on the treated side by eight weeks, strengthening causal inference beyond single-arm data [35].

Table 1: Representative intradermal botulinum toxin injection parameters reported for oily skin, enlarged pores, and acne vulgaris [27,28,29,30].			
Series	Toxin / dilution	Injection plane	Total dose per session
[27]	OnabotulinumtoxinA, diluted saline	Superficial intradermal	~20-30 U, full face
[28]	OnabotulinumtoxinA, 100 U/4 mL	Superficial intradermal	~30-40 U, full face
[29]	OnabotulinumtoxinA, 100 U/8 mL	Superficial intradermal microdroplet	~50-60 U, full face
[30]	IncobotulinumtoxinA, diluted saline	Superficial intradermal	~30 U, target zones

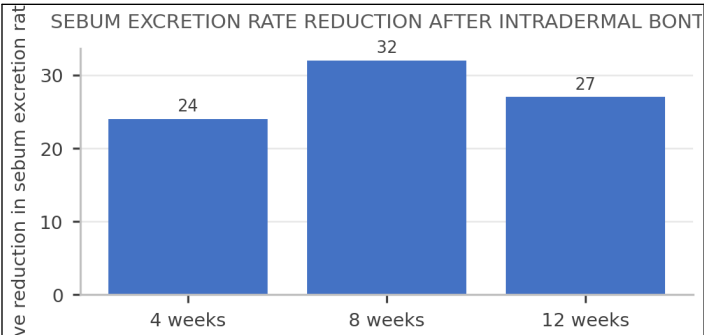


Figure 1: Mean relative reduction in sebum excretion rate from baseline at four and twelve weeks after a single intradermal botulinum toxin type A treatment session, pooled from prospective and split-face series in seborrheic and acne-prone skin [28,27,29,35].

More recent work targeting an acne-specific population has extended these findings. Sayed and colleagues, treating patients with mild to moderate acne and coexisting oily, large-pored skin, reported significant reductions in sebum excretion rate, oiliness score, and improved satisfaction at four and twelve weeks, correlating with baseline sebum output [29]. Kang and colleagues' pilot study similarly found significant sebum reductions alongside improved seborrhea severity grading, sustained through twelve weeks [30]. These series consistently show a 20-40% relative reduction from baseline sustained for two to four months, broadly consistent with BoNT-A's known duration of effect [26,25].

6. Clinical outcomes: pore size and skin texture

Because pore diameter tracks sebaceous gland activity closely, most series measuring sebum reduction also report a parallel, smaller-magnitude reduction in pore size on standardized imaging [14,15]. Shah documented visibly narrowed pores at eight weeks, attributed to reduced follicular distension from lower sebaceous output rather than any structural change to the follicular ostium [27], and Sayed and colleagues found a statistically significant reduction in mean pore area at twelve weeks, correlated with the concurrent sebum reduction [29].

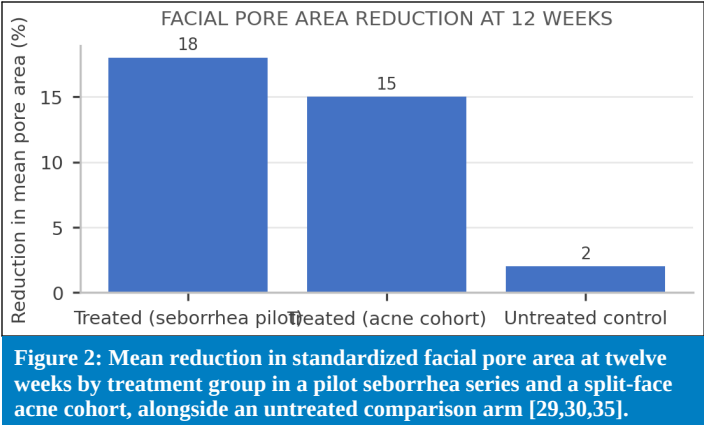
Patient-reported outcomes corroborate the imaging data: global aesthetic improvement scales and satisfaction questionnaires consistently rank pore size and texture improvement among the most valued outcomes, often exceeding satisfaction with lesion clearance, since oiliness and pores are the daily, visible concerns that motivate many patients to seek treatment [30,28]. This suggests intradermal BoNT-A is best suited to patients whose chief complaint is seborrhea and coarse pores rather than nodulocystic disease, for whom systemic therapy remains first-line [21,20].

7. Clinical outcomes: inflammatory lesions and scarring risk

Evidence on inflammatory lesion counts is more limited, reflecting smaller samples and inconsistent counting methodology. Sayed and colleagues, the largest series to report a formal severity grade, found a significant reduction in total lesion count and Global Acne Grading System score at twelve weeks, though the effect was smaller than for sebum output or pore size, and the trial was not powered against a standard topical regimen [29]. Kang and colleagues' pilot data similarly suggested fewer inflammatory lesions at follow-up, though sample size limited statistical power [30].

Table 2: Summary of clinical endpoints reported across the principal intradermal botulinum toxin acne/seborrhea series [29,30,28,27].

Endpoint	Direction of change	Typical follow-up	Consistency across series
Sebum excretion rate	Decreased	4-12 weeks	Consistent, largest effect size
Facial pore area	Decreased	8-12 weeks	Consistent, moderate effect size
Inflammatory lesion count	Decreased	12 weeks	Reported in fewer series, smaller effect
Patient satisfaction / GAIS	Improved	8-12 weeks	Consistent



Plausible contributors to any lesion benefit are reduced sebum substrate for C. acnes and follicular occlusion, plus the anti-neurogenic-inflammatory effect of BoNT-A on substance P and calcitonin gene-related peptide release described above [24,25]. Because scarring risk correlates with lesion depth and duration rather than count alone, no series has yet followed patients long enough, with standardized scar-grading instruments, to establish whether these modest reductions lower scarring outcomes, a gap the field still needs to close [19,36]. Until such data exist, BoNT-A should be regarded as a seborrhea- and pore-directed adjunct rather than a scar-prevention strategy.

8. Safety, tolerability, and adverse events

The adverse-event profile mirrors cosmetic BoNT-A more broadly: transient injection-site bruising, mild erythema, and pinpoint bleeding are common but self-limited, resolving within 24-72 hours [28,27,32]. Because the microdroplet technique avoids deep muscular targeting, motor complications such as brow ptosis or asymmetric

smile, well recognized elsewhere in cosmetic BoNT-A, are rare when injectors keep to superficial depth and avoid the periorbital and perioral zones [33,34,10]. No acne-indication series has reported a serious systemic adverse event, consistent with the low total doses used relative to those associated with distant toxin spread in other indications [23,9].

Session discomfort, driven mainly by needle-stick sensation across many injection points, is mild to moderate and improved with topical anesthesia, and does not appear to limit repeat treatment uptake, with most cohorts reporting high retreatment rates at three to six months [31,11]. Cost and the need for a trained injector remain practical barriers to wider adoption outside specialist aesthetic practices relative to inexpensive topical standards of care [7,5].

9. Positioning relative to conventional acne therapy

Intradermal BoNT-A is not proposed as a replacement for guideline-directed topical or antibiotic therapy in inflammatory acne, nor for hormonal or isotretinoin therapy in more severe disease [4,37]. Its evidence base instead supports a narrower, adjunctive role for patients whose dominant residual complaint is oiliness, enlarged pores, and coarse texture, or who cannot tolerate systemic sebosuppressive options [6,22]. Several authors have proposed combining low-dose intradermal BoNT-A with a standard topical regimen, reasoning that the two act through largely non-overlapping mechanisms, though comparative and combination trials remain scarce relative to the pilot data available today [16,26].

10. Conclusion

Published evidence, still confined to small open-label series, one split-face comparison, and a handful of prospective cohorts, consistently shows that superficial intradermal botulinum toxin reduces sebum excretion rate and facial pore size in acne vulgaris and seborrhea-predominant skin, with a more modest and less consistently demonstrated benefit on inflammatory lesion

counts [28,27,29,30]. The mechanistic rationale, cholinergic blockade of sebocyte lipogenesis reinforced by an anti-neurogenic-inflammatory effect on perifollicular sensory nerve terminals, is biologically coherent and supported by in vitro work, even though large randomized trials with standardized lesion-counting and long-term scar follow-up have not yet been performed [13,24,25]. Given its favorable safety profile at the low doses used for this indication, intradermal botulinum toxin is best regarded as a well-tolerated adjunct for sebum- and pore-driven concerns in acne-prone patients, positioned alongside rather than in place of guideline-directed acne therapy, pending the larger comparative trials the field still needs [4,26,11].

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