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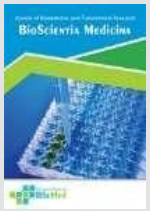

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Hydrostatic Pressure versus Passive Diffusion: A Split-Face Comparative Analysis of Intradermal Injection and Microneedling-Assisted Delivery of Botulinum Toxin Type A for Facial Pore Refinement

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A B S T R A C T

Background: Enlarged facial pores, medically termed dilated pilosebaceous follicles, represent a prevalent aesthetic concern driven by seborrhea, follicular hypertrophy, and loss of perifollicular elasticity. Microbotox, the intradermal administration of dilute OnabotulinumtoxinA (BoNT-A), targets these mechanisms through sebosuppression and arrector pili inhibition. However, the optimal delivery vehicle—active intradermal injection versus passive microneedling-assisted transport—remains debated regarding clinical delivery efficiency. **Case presentation:** A 23-year-old female with Fitzpatrick Skin Type IV, severe pore enlargement (Kim's Score 5), and seborrhea participated in a split-face comparative study. The right cheek received standard intradermal microdroplet injections of BoNT-A (20 U diluted in 1.0 mL saline). The left cheek underwent automated microneedling at a depth of 2.0 mm immediately followed by topical application of the same BoNT-A solution. Evaluation was performed at baseline, Day 7, and Day 14 using blinded clinical scoring and digital dermoscopic analysis. At Day 14, the intradermal injection side demonstrated superior pore reduction (Kim's Score 5 to 3) compared to the microneedling side (Score 5 to 4). Digital quantification confirmed a 45% reduction in mean pore diameter on the injected side versus 18% on the microneedling side. While both modalities effectively reduced sebum scores to 1, the microneedling side exhibited delayed pore refinement, likely attributed to post-traumatic edema and the wash-out effect of blood flow antagonizing passive diffusion. **Conclusion:** Direct intradermal injection provides superior clinical delivery efficiency for BoNT-A, resulting in more rapid and significant pore contraction. Microneedling-assisted delivery, particularly at depths inducing vascular injury, acts as a secondary adjunct for textural remodeling but is inferior for immediate pharmacological delivery of large-molecule toxins.

1. Introduction

The relentless pursuit of flawless skin texture—characterized by high luminosity, uniform tone, and minimal topographic irregularity—has become a defining feature of contemporary aesthetic dermatology. In an era driven by high-definition digital media, the demand for procedures that refine cutaneous topography has surged.[1] Among the myriad of cosmetic complaints encountered in clinical practice, enlarged facial pores represent a ubiquitous and distressing concern. Clinically defined as dilated openings of the pilosebaceous follicles visible to the naked eye, these ostia are medically benign yet hold profound aesthetic significance. While they do not pose a physiological threat, their enlargement is frequently interpreted by the observer as a hallmark of cutaneous aging, poor hygiene, or solar damage. This stigma is particularly pronounced in Asian

populations, where cultural beauty standards heavily prioritize smooth, porcelain-like skin texture.[2] Consequently, the presence of enlarged pores often leads to measurable psychosocial impairment, driving patients to seek dermatological intervention to ameliorate the perception of unclean or masculinized skin.

To address this concern effectively, one must first deconstruct the complex pathophysiology of the pilosebaceous unit. Pore enlargement is not a singular event but the result of a multifactorial triad: excessive sebum production (seborrhea), decreased structural integrity of the perifollicular dermis, and hypertrophy of the hair follicle volume.[3] The sebaceous gland, an integral component of this unit, operates under distinct neuroendocrine control. Sebum output is primarily regulated by androgens, specifically dihydrotestosterone (DHT), which binds to nuclear receptors on sebocytes to trigger cellular proliferation and lipid synthesis. When sebum production becomes excessive—whether due to hormonal fluctuations, genetics, or environmental stressors—the constant, high-volume flow of lipids exerts outward hydrostatic pressure on the infundibulum. This chronic flow mechanically dilates the canal, much like water widening a riverbed. Simultaneously, the structural scaffold surrounding the pore undergoes degradation. The perifollicular dermis acts as a corset, providing a passive contraction force that keeps the pore orifice tight. With the onset of intrinsic aging and photoaging, the collagen and elastin fibers in this region fragment and diminish. As this dermal support weakens, the skin loses its tensile strength and elasticity, causing the pore walls to slacken and the aperture to appear more patulous and oval-shaped. This loss of elasticity creates a vicious cycle where the pore is unable to recoil against the pressure of sebum outflow, leading to permanent dilation.

Historically, the therapeutic landscape for enlarged pores has been dominated by modalities that target either the epidermal surface or the sebaceous gland indirectly. Chemical peels utilizing salicylic or glycolic acid aim to exfoliate the stratum corneum and dissolve keratinous plugs, while topical retinoids accelerate cell turnover and normalize keratinization.[4] In the realm of energy-based devices, fractional CO_2 lasers and radiofrequency microneedling have been employed to induce thermal injury, thereby stimulating neocollagenesis to tighten the perifollicular dermis. However, these conventional approaches are not without limitations. Ablative lasers and aggressive chemical resurfacing are frequently associated with significant downtime, discomfort, and a heightened risk of post-inflammatory hyperpigmentation (PIH), particularly in patients with Fitzpatrick skin types IV through VI. Furthermore, while these methods address the structural component of pore enlargement, their ability to control the neuroendocrine driver—the sebaceous gland itself—is often variable or temporary.

In response to these limitations, a paradigm shift has occurred with the introduction of the intradermal administration of Botulinum Toxin Type A (BoNT-A), a technique colloquially known as Microbotox, Mesobotox, or Intradermal Botox.[5] This technique represents a refined application of the neurotoxin, distinct from its traditional on-label use for dynamic rhytids where the target is the neuromuscular junction of skeletal muscles. In the context of Microbotox, highly diluted concentrations of BoNT-A are deposited directly into the dermis rather than the underlying muscle. The mechanism of action for Microbotox is elegant and targeted. BoNT-A functions by cleaving the SNAP-25 protein, effectively inhibiting the release of acetylcholine from presynaptic vesicles. While traditionally associated with muscle paralysis, acetylcholine is also a critical neurotransmitter at the neuroglandular junction of the sebaceous glands. Sebocytes express muscarinic acetylcholine receptors (specifically the M3 subtype), and their activation stimulates differentiation and sebum secretion. By blocking this cholinergic signaling, intradermal BoNT-A induces profound sebosuppression, thereby reducing the volume of lipid flow that actively dilates the pore. Additionally, the toxin targets the arrector pili muscle, a microscopic smooth muscle attached to

the follicle. It is hypothesized that the contraction of this muscle exerts mechanical traction on the follicular wall; thus, inducing flaccid paralysis of the arrector pili may allow the pore orifice to relax and flatten, contributing to a smoother cutaneous topography. Some evidence further suggests that BoNT-A may modulate fibroblast activity, potentially enhancing skin tensile strength and creating a lifting effect that compresses the pore.

Despite the growing popularity and established pharmacological rationale of Microbotox, a significant controversy persists regarding the optimal method of delivery. The efficacy of the treatment is inextricably linked to the ability of the toxin to reach its target receptors in the reticular dermis.[6] The standard, gold standard technique involves multiple serial intradermal injections (nappage technique). This method ensures precise deposition of the toxin at the desired depth. However, it is a labor-intensive process requiring hundreds of needle pricks, which can be technically demanding for the practitioner and painful for the patient. The trauma associated with serial injection also carries a risk of bruising and diffusion irregularities if not performed with high precision.[7]

Seeking a more efficient and patient-friendly alternative, clinicians have increasingly turned to microneedling (collagen induction therapy) as a transdermal drug delivery system (TDDS). Microneedling involves the use of automated devices to create thousands of temporary micro-channels through the stratum corneum and into the dermis. Theoretically, these channels serve as conduits that bypass the skin's primary physical barrier, facilitating the passive transport of topically applied BoNT-A down to the sebaceous glands. Proponents of this method argue that it offers a synergistic benefit: the delivery of the toxin combined with the wound-healing response triggered by the needles, which stimulates the release of platelet-derived growth factor (PDGF) and transforming growth factor-beta (TGF-β) to induce collagen remodeling.[8]

However, the assumption that microneedling acts as an efficient delivery vehicle for BoNT-A remains under-scrutinized in comparative trials. BoNT-A is a large molecule with a molecular weight of approximately 150 kDa (core protein) to 900 kDa (complex), making it difficult to penetrate the dermis via passive diffusion alone.[9] Critical questions regarding bioavailability remain unanswered. Does the physical barrier of the skin, even when disrupted by micro-channels, prevent adequate toxin absorption compared to direct injection?. Furthermore, does the exudation of blood and interstitial fluid caused by the trauma of microneedling create an outward pressure gradient that effectively washes away the topically applied toxin before it can reach the sebocytes?. Conversely, does the direct intradermal injection provide a superior, hydrostatic pressure-driven blockade of sebaceous activity that outweighs the convenience of microneedling?. There is a paucity of literature directly comparing the clinical delivery efficiency of these two modalities in a controlled manner. Most existing studies evaluate one method in isolation or combined with other agents, making it difficult to isolate the variable of delivery method. This gap in knowledge leaves clinicians without clear, evidence-based guidelines on whether to prioritize the precision of injection or the synergy of microneedling for pore refinement.[10]

This study aims to bridge this critical gap in dermatological literature by evaluating and comparing the clinical and dermoscopic outcomes of two distinct Microbotox delivery modalities—intradermal injection versus microneedling-assisted delivery—within a single biological system. By utilizing a rigorous split-face design, this research controls for systemic variables such as genetics, hormonal status, and environmental exposure, allowing for a direct comparison of local delivery efficiency. The novelty of this study lies in its specific focus on the delivery efficiency of the toxin in the context of pore pathophysiology. Unlike previous studies that may focus solely on subjective improvement, this research incorporates dermoscopic validation to quantify changes in pore diameter and sebum output. Furthermore, it critically examines the impact of

delivery method on the rapidity of onset and the magnitude of sebosuppression. This investigation seeks to determine whether the passive diffusion model of microneedling is clinically equivalent to the active placement model of injection, thereby providing clinicians with an evidence-based recommendation for optimizing pore refinement protocols and establishing a gold standard for the administration of Microbotox.

2. Case Presentation

Written informed consent was obtained from the patient for the publication of this case report, including the use of clinical photographs and dermoscopic imagery, in accordance with the Declaration of Helsinki. A 23-year-old female patient of Asian descent presented to the Dermatology and Venereology outpatient clinic seeking evaluation and management for severe textural irregularities of the facial skin. Classification of her skin phototype revealed Fitzpatrick Skin Type IV (moderate brown, tans easily, burns minimally), a clinically significant factor given the heightened propensity for post-inflammatory hyperpigmentation (PIH) in this demographic following procedural interventions. Her primary aesthetic concern was the presence of large, visible holes localized to the centrofacial region, specifically the medial cheeks and nasal ala, which she felt compromised her facial aesthetics. Concurrently, she reported intractable facial oiliness, clinically consistent with severe seborrhea, which contributed to a shiny, unkempt appearance necessitating frequent cosmetic management.

Upon detailed interrogation, the patient reported a chronic course of enlarged facial pores spanning approximately eight years. The onset of the condition coincided with menarche and the onset of puberty, highlighting the probable androgen-driven pathogenesis of her follicular enlargement. While the condition had been present for nearly a decade, she noted a progressive exacerbation in the severity of the pore size and sebum output over the preceding two years. The patient described her skin as constantly greasy, a symptom of seborrhea oleosa that required the use of blotting papers or powder multiple times daily to maintain a matte appearance. This chronic seborrhea not only contributed to the physical dilation of the pores via continuous lipid flow but also served as a source of significant psychosocial distress and cosmetic anxiety.

The patient's dermatologic background was significant for inflammatory acne vulgaris during adolescence. While the acute inflammatory phase of the disease had resolved without the need for systemic isotretinoin, the sequelae remained evident. She noted residual textural irregularities that she distinguished from the pores themselves. This scarring was identified as a mix of mild atrophic scars, specifically the ice-pick and boxcar variants, which frequently coexist with enlarged pores in patients with a history of acne. Crucially for the safety profile of the proposed microneedling intervention, the patient explicitly denied any personal or familial history of keloid formation or hypertrophic scarring.

An assessment of the patient's lifestyle revealed extrinsic factors likely exacerbating her intrinsic genetic predisposition. Her skincare regimen was notably minimal and insufficient for her skin type, consisting solely of an over-the-counter foaming facial cleanser utilized twice daily. She reported a complete absence of corrective topical agents; she did not utilize toners, serums, moisturizers, or—most critically—photoprotection (sunscreen). The lack of routine exfoliation or retinoid use likely contributed to follicular hyperkeratosis, where retention of dead skin cells at the pore orifice further obstructs and dilates the canal. Furthermore, a dietary review indicated a high consumption of high-glycemic-index foods, with a specific predilection for refined sugars and sweet foods. This dietary pattern is clinically relevant as hyperglycemic states trigger the insulin/insulin-like growth factor-1 (IGF-1) cascade, a known stimulant of sebocyte proliferation and lipogenesis. The patient was a non-smoker and denied alcohol consumption, ruling out these factors as contributors to her cutaneous condition. The patient represented a naive therapeutic candidate. She was not currently prescribed any

systemic medications, including oral hormonal contraceptives (which could alter sebum levels) or oral isotretinoin. Furthermore, she was not utilizing any topical pharmacological agents such as antibiotics or retinoids, ensuring that the baseline assessment reflected her unmedicated physiological state. She reported no known drug, food, or environmental allergies.

A rigorous and comprehensive physical examination was conducted under standard ambient lighting followed by magnified illumination to characterize the cutaneous topography. The patient appeared well-nourished and in good general health, with stable vital signs. The facial examination revealed a symmetrical, bilateral distribution of pathology, validating the suitability of a split-face study design. The pathology was most pronounced in the T-zone and medial infraorbital regions. The cutaneous surface exhibited marked topographic heterogeneity. The skin appeared pebbled and uneven, a presentation attributed to the coalescence of dilated follicular ostia and mild atrophic acne scarring. The scarring was predominantly of the ice-pick (narrow, deep) and boxcar (broad, rectangular) subtypes, creating a distinct negative vertical relief compared to the surrounding skin. Clinical signs of seborrhea were profound. The forehead, nose, chin, and medial cheeks exhibited a high-gloss shine indicative of excessive sebum excretion rates. While open and closed comedones were not the primary feature, the follicular ostia appeared congested. No active inflammatory papules, pustules, or cysts were observed, allowing for immediate procedural intervention without the risk of spreading infection (Figure 1). The background skin tone was uneven, punctuated by multiple ill-defined, hyperpigmented macules. These lesions were consistent with post-inflammatory hyperpigmentation (PIH) resulting from prior acne excoriations, confirming the reactive nature of her melanocytes. To minimize subjectivity and ensure rigorous monitoring, standardized scoring systems were employed by a blinded evaluator to establish baseline metrics. Utilizing the scale established by Kim et al. (0-6), the patient's condition was classified as severe; Right Cheek: Score 5; Left Cheek: Score 5; Interpretation: A score of 5 represents pores that are visually prominent and widely distributed, constituting a significant aesthetic disfigurement. A visual assessment scale (0-3) was used to quantify oiliness; Right Cheek: Score 3; Left Cheek: Score 3; Interpretation: A score of 3 denotes excessive oiliness, characterizing the skin as distinctly greasy to the touch and visually reflective.

To complement the clinical grading with sub-clinical detail, non-contact polarized dermoscopy was performed. This allowed for the visualization of the follicular architecture free from surface glare. The captured images were subsequently processed using ImageJ software (National Institutes of Health) to provide objective morphometric data regarding pore diameter; (1) Right Cheek: The dermoscopic field of view was dominated by numerous shallow holes, representing the dilated infundibula of the hair follicles. A hallmark finding was the abundance of yellow dots. Pathologically, these represent distended follicular openings filled with oxidized sebum and keratinous debris (micro-comedones), confirming the role of blockage in her pore expansion. The mean pore diameter was measured at 0.62 ± 0.08 mm, a value significantly higher than the average pore size typically reported in healthy skin (<0.2 mm), confirming the severity of follicular hypertrophy; (2) Left Cheek: The dermoscopic findings were mirror-identical to the right, confirming anatomical symmetry. The yellow dot density was equally high, and the mean pore diameter was measured at 0.64 ± 0.07 mm. The significant follicular prominence observed dermoscopically correlated perfectly with the clinical Kim's score of 5, establishing a robust baseline for comparing the therapeutic interventions.

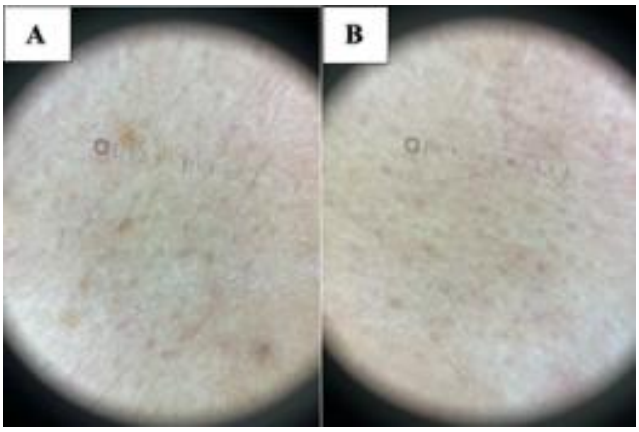

Figure 1. Dermoscopic findings before treatment. (A) Right cheek; (B) Left cheek.

Table 1. Summary of Clinical Findings on Admission

Baseline patient demographics, dermatologic history, and split-face assessment parameters.

PARAMETER	CLINICAL DETAIL
1. PATIENT PROFILE & DEMOGRAPHICS	
Demographics	23-year-old Female; Asian Descent; Fitzpatrick Skin Type IV .
Chief Complaint	Large, visible holes on medial cheeks/nose and excessive facial oiliness (Seborrhea).
Duration	~8 years (onset at puberty); progressive worsening over last 2 years.
2. DERMATOLOGIC HISTORY & LIFESTYLE	
Dermatologic History	History of inflammatory acne vulgaris (teenage years). **Scarring:** Residual mild atrophic scars (Ice-pick and Boxcar types). **Negatives:** No keloids or hypertrophic scarring.
Risk Factors (Exposome)	**Diet:** High Glycemic Index (frequent refined sugars). **Skincare:** Minimal (OTC cleanser only). No toner, serum, or moisturizer. **Photoprotection:** No regular sunscreen use .
Medications	Treatment Naive (No oral contraceptives, isotretinoin, or topical retinoids/antibiotics).
3. PHYSICAL EXAMINATION	
Cutaneous Morphology	**Texture:** Uneven, pebbled surface due to dilated ostia and scarring. **Seborrhea:** Significant shine (T-zone and medial cheeks). **Pigmentation:** Ill-defined post-inflammatory hyperpigmentation (PIH) macules.
4. BASELINE QUANTITATIVE SCORING (SPLIT-FACE)	
Right Cheek (Injection Side)	• **Enlarged Pore Score (Kim et al.):** Score 5 (Severe) • **Sebum Score:** Score 3 (Excessive)
Left Cheek (Microneedling Side)	• **Enlarged Pore Score (Kim et al.):** Score 5 (Severe) • **Sebum Score:** Score 3 (Excessive)
5. BASELINE DERMOSCOPY	
Findings	Numerous shallow holes (dilated infundibula). Abundant **Yellow Dots** indicating sebum accumulation and keratinous plugging.
Pore Diameter	Range: **0.5 mm to 1.5 mm** (Symmetrical distribution).

The patient provided written informed consent to participate in a split-face comparative treatment protocol. This study design is considered the gold standard in dermatological research for topical and localized interventions, as it effectively neutralizes systemic confounding variables—such as hormonal fluctuations, genetic predisposition, circadian rhythms, and environmental exposures—allowing for a precise, side-by-side evaluation of local delivery efficiency (Table 2).

To ensure an aseptic field and maximize therapeutic uptake, a rigorous pre-procedural protocol was implemented. The facial skin was first subjected to thorough cleansing to solubilize and remove the hydrolipid film, environmental debris, and transient microbial flora. This degreasing step is critical, particularly in seborrheic patients, as surface lipids can impede the penetration of topical anesthetics and active agents. Following cleansing, a topical anesthetic cream containing Lidocaine 10.56% was applied to the entire facial surface. To facilitate transdermal penetration of the anesthetic, the area was covered with an occlusive dressing (plastic wrap) for 45 minutes. Occlusion hydrates the stratum corneum, swelling the corneocytes and increasing the permeability of the skin barrier, thereby ensuring optimal anesthesia and minimizing procedural discomfort (VAS scores). Upon removal of the anesthetic and occlusion, a two-step disinfection protocol was employed. The skin was first wiped with 70% isopropyl alcohol to remove anesthetic residue, followed by chlorhexidine gluconate to provide broad-spectrum antimicrobial prophylaxis. This dual-step approach is vital to prevent the translocation of surface bacteria into the deep dermis during needle penetration.

The therapeutic agent utilized was OnabotulinumtoxinA (BoNT-A), supplied in a standard 100-Unit vial. The reconstitution protocol for microbotox differs significantly from the standard on-label dilution used for neuromuscular blockade in dynamic rhytids. The objective of microbotox is to target the superficial muscarinic receptors of the sebaceous glands and the dermal arrector pili muscles, rather than the deep muscles of facial expression. Therefore, a hyper-dilution technique was employed to facilitate widespread diffusion within the dermal plane. A total of 20 Units of BoNT-A were withdrawn for the treatment. These 20 Units were reconstituted in 1.0 mL of 0.9% sterile physiological saline (NaCl). This admixture yielded a final concentration of 2 Units per 0.1 mL. This high volume-to-unit ratio is strategic; the increased volume acts as a carrier, allowing the practitioner to cover a larger surface area (the medial cheeks) and ensuring that the hydrostatic pressure of the injection spreads the active molecule horizontally across the dermo-epidermal junction. The right cheek was designated as the active control side, utilizing the traditional manual injection technique which prioritizes precision and guaranteed depth. A geometric grid was drawn on the medial cheek using a surgical marker, with injection points spaced equidistantly at 1.0 cm intervals. This spacing was calculated to allow the diffusion halos of the toxin to overlap slightly, creating a confluent field of effect. A 1.0 mL tuberculin syringe fitted with a fine-gauge 30G needle was employed. At each marked point, a micro-aliquot of approximately 0.05 mL (containing 1 Unit of BoNT-A) was administered. The needle was inserted at an acute angle of 30 degrees relative to the skin surface, penetrating to a depth of approximately 2.0 mm. This depth is critical; it targets the reticular dermis where the sebaceous glands reside, avoiding the deeper subcutaneous plane where the zygomaticus major and minor muscles create facial expression. The injection was performed slowly until a distinct, blanched, superficial papule (wheal) appeared. This visual endpoint confirms that the solution has been deposited intradermally. The formation of the wheal creates immediate hydrostatic pressure, physically dissecting the collagen bundles and forcing the fluid to interface directly with the target receptors.

The left cheek was treated with a hybrid protocol combining mechanical remodeling with pharmacological delivery. An automated electric

microneedling device was utilized. A needle depth of 2.0 mm was selected. While standard transdermal drug delivery systems (TDDS) often utilize shallower depths (0.5–1.0 mm), the 2.0 mm depth was chosen to address the patient's concurrent atrophic scarring. This depth penetrates through the epidermis and well into the reticular dermis, inducing localized vascular injury and triggering the wound-healing cascade. The remaining BoNT-A solution (approximately 10 Units in 0.5 mL) was drawn into a syringe for topical application. The skin was held taut to ensure uniform needle penetration. The device was glided over the treatment area in multiple vectors—horizontal, vertical, and diagonal—to maximize the density of micro-channels. The clinical endpoint was defined as the appearance of uniform pinpoint bleeding (petechiae) and erythema, indicating successful breach of the vascularized dermis. Immediately upon cessation of needling, while the micro-channels remained patent, the BoNT-A solution was dripped onto the skin surface. The area was gently massaged to facilitate passive diffusion of the macromolecule through the channels and into the dermis. This method relies on gravity and capillary action rather than the active hydrostatic pressure used on the right side. To mitigate the risk of infection in the open micro-channels, a topical antibiotic cream (Fusidic acid 2%) was applied to both treated areas. The patient was discharged with specific instructions to maintain the integrity of the treatment: avoidance of facial washing for 4 hours to prevent washing away the topical toxin (on the left) or disturbing the injection sites (on the right), and avoidance of heavy makeup for 24 hours to prevent the introduction of foreign pigment into the open channels.

On post procedural phase (day 0), the immediate response highlighted the distinct physical trauma profiles of the two modalities. The patient reported the procedure as tolerable, rating the pain as mild (2–3 on the VAS). Bilateral erythema (redness) was the universal finding. However, the morphological presentation differed: the right cheek (injection) displayed the characteristic cobblestone appearance of saline-filled papules, which typically resolve within hours as the fluid absorbs. In contrast, the left cheek (microneedling) exhibited diffuse edema (swelling) consistent with the inflammatory response to multiple needle penetrations.

By 48 hours, the acute nociceptive response had vanished (VAS 0). On the right cheek, the injection sites had largely healed, presenting only with mild residual erythema and faint pinpoint ecchymosis (bruising) at the puncture sites. Left cheek, the trauma from the 2.0 mm microneedling resulted in a more sustained recovery. Pronounced erythema persisted, accompanied by mild desquamation. This peeling is a hallmark of the re-epithelialization process following mechanical exfoliation. At this early stage, no visible reduction in pore size was noted. This is consistent with the known pharmacodynamics of Botulinum Toxin, which typically requires 3–7 days to inhibit acetylcholine release effectively. Furthermore, any structural changes were likely masked by post-procedural inflammation and edema.

One week post-procedure marked the onset of therapeutic efficacy (Figure 2). The patient reported a noticeable reduction in facial oiliness. Clinically, the skin appeared less greasy, with the Sebum Score decreasing from 3 (excessive) to 2 (moderate) on both sides. A synchronous improvement was observed. The Visual Pore Score decreased from 5 to 4 on both the right and left cheeks. Magnified analysis confirmed a reduction in the visibility of yellow dots (keratin/sebum plugs), validating that the blockade of sebaceous activity was initiating the clearance of follicular congestion.

At the two-week mark, the maximal effect of the BoNT-A was realized, revealing a significant divergence in efficacy between the two delivery methods. On the right cheek (intradermal injection), this side exhibited the superior outcome. The skin texture appeared tightened with a distinct matte finish, reflecting profound sebosuppression. The Sebum Score dropped to 1 (mild oiliness). The Visual Pore Score improved significantly to 3.

Figure 2. Follow-up photographs before and after treatment. A. Right cheek treated with intradermal microbotox. B. Left cheek treated with microneedling.

Dermoscopic quantification provided rigorous validation of this improvement: the mean pore diameter was reduced to 0.34 ± 0.05 mm (range 0.02–1.0 mm). This represents a marked 45% reduction from baseline. The follicular borders appeared softened and less defined, blending seamlessly into the surrounding skin. This confirms that direct injection maximizes bioavailability, delivering a high concentration of toxin to the target receptors. On the left cheek (microneedling-assisted); this side showed improvement but lagged behind the injection side in specific parameters. While the Sebum Score also decreased to 1, indicating that enough toxin penetrated to affect oil production, the structural reduction of the pores was less dramatic. The Visual Pore Score remained at 4. The mean pore diameter measured 0.52 ± 0.06 mm (range 0.05–1.0 mm). This represents only an 18% reduction from baseline. Despite the inferior pore contraction, the left side exhibited superior improvement in the *texture* of the atrophic acne scars. This suggests that while the passive diffusion of toxin was less effective for shrinking pores than direct injection, the mechanical trauma of the microneedling successfully triggered the wound-healing response necessary for scar remodeling. In summary, the clinical course demonstrated that while both modalities are safe and effective for sebosuppression, the intradermal injection technique provides a statistically superior and more rapid reduction in pore caliber (45% vs. 18%) due to the hydrostatic assurance of drug delivery. The microneedling approach, heavily influenced by the 2.0 mm depth, acted primarily as a textural resurfacing tool with secondary pharmacological benefits.

Table 2. Diagnosis, Treatment Protocol, and Clinical Outcomes
Comprehensive summary of split-face methodology, post-procedural course, and Day 14 endpoints.

1. FINAL DIAGNOSIS		
Primary Diagnosis: Severe Enlarged Facial Pores (Kim's Score 5) with Seborrhea Oleosa. **Secondary Diagnosis:** Mild Atrophic Acne Scarring (Ice-pick & Boxcar type). **Skin Phenotype:** Fitzpatrick Skin Type IV (High risk for PIH).		
2. THERAPEUTIC INTERVENTION (SPLIT-FACE PROTOCOL)		
PARAMETER	**RIGHT CHEEK (ACTIVE CONTROL)**	**LEFT CHEEK (COMPARATIVE)**
Modality	**Intradermal Injection** *Microbotox Technique*	**Microneedling-Assisted** *Automated Needling + Topical Application*
Dosage & Dilution	20 Units / 1.0 mL Saline (2 Units per 0.1 mL concentration)	~10 Units (Topical Drip) Applied immediately post-needling
Depth & Technique	**Depth:** 2.0 mm (Intradermal) **Technique:** Manual Serial Puncture (1 cm apart) **Hydrostatic Pressure**	**Depth:** 2.0 mm (Deep Dermal) **Technique:** Vertical/Horizontal Passes **Passive Diffusion**
3. CLINICAL COURSE & SAFETY PROFILE		
Day 0 (Immediate)	Transient wheals (resolved < 4 hrs). Mild erythema. Pain: VAS 2-3.	Pinpoint bleeding & crusting. Significant edema (swelling). Pain: VAS 2-3.
Day 2 (Recovery)	Minimal pinpoint ecchymosis. **Pain Resolved**	Pronounced erythema. Visible desquamation (peeling). **Longer Downtime**
Day 7 (Onset)	**Sebum:** Reduced (Score 3 → 2). **Pore:** Visual improvement (Score 5 → 4).	**Sebum:** Reduced (Score 3 → 2). **Pore:** Visual improvement (Score 5 → 4). Texture appearing smoother.
4. OUTCOME AT DAY 14 (PRIMARY ENDPOINT)		
Sebum Score (0-3 Scale)	**Score 1** (Mild) **Effective Blockade**	**Score 1** (Mild) **Effective Blockade**
Pore Score (Kim's 0-6 Scale)	**Score 3** Significant Reduction from Baseline (5).	**Score 4** Modest Reduction from Baseline (5).
Dermoscopic Analysis	Mean Diameter: **0.34 mm** Reduction: **45% Decrease** Morphology: Collapsed borders, matte appearance.	Mean Diameter: **0.52 mm** Reduction: **18% Decrease** Morphology: Reduced yellow dots, patent structure.
Secondary Benefits	Tightening effect. Superior pharmacologic delivery.	**Texture Improvement:** Superior softening of atrophic acne scars. *(Likely due to edema/wound healing)*

3. Discussion

This split-face study provides clinical and dermoscopic evidence comparing two methods of delivering Botulinum Toxin Type A for the treatment of enlarged facial pores. The results indicate that while both techniques are effective, intradermal injection yields a superior and more rapid reduction in pore size compared to microneedling-assisted delivery.[11]

To understand the efficacy of Microbotox, one must consider the anatomy of the pilosebaceous unit. The sebaceous gland is an exocrine gland whose activity is regulated by hormonal and neuroendocrine factors.[12] Crucially, sebocytes express muscarinic acetylcholine receptors (specifically M3). Acetylcholine released from cutaneous nerve endings binds to these receptors, stimulating sebocyte differentiation and sebum secretion. BoNT-A acts by cleaving the SNAP-25 protein, preventing the fusion of synaptic vesicles with the presynaptic membrane, thereby blocking the release of acetylcholine. By blocking cholinergic signaling at the sebaceous gland, BoNT-A reduces sebum production. Since the volume of sebum contributes to the physical dilation of the pore canal, reduced flow leads to a passive reduction in pore diameter. The arrector pili muscle inserts near the sebaceous gland. It has been hypothesized that contraction of this muscle exerts mechanical traction on the follicle, keeping the pore open. BoNT-A induces flaccid paralysis of this microscopic muscle, potentially allowing the pore orifice to relax and close. Some studies suggest that BoNT-A may modulate fibroblast activity, potentially tightening the dermal matrix, which creates a lifting effect that compresses the pore.[13]

The core finding of this study—that injection is superior to microneedling for pore reduction—can be explained by clinical delivery efficiency and depth of delivery. Intradermal injection (the right side); this technique ensures that 100% of the calculated dose is delivered directly into the reticular dermis, where the sebaceous glands and arrector pili muscles reside.[14] The creation of a wheal generates hydrostatic pressure, forcing the toxin to diffuse horizontally through the interstitial fluid to reach the target receptors. The result is a high local concentration of the drug, leading to a profound blockade of cholinergic activity. This explains the significant drop in Sebum Score (3 to 1) and the dramatic reduction in pore diameter observed on the right side.[15]

Microneedling creates micro-channels that bypass the stratum corneum, the primary barrier of the skin. However, this is a passive delivery system. A critical methodological consideration in this study was the use of a 2.0 mm needle depth. While standard transdermal drug delivery protocols often recommend 0.6 mm to 1.0 mm depths to breach the epidermal barrier without inducing heavy bleeding, this study utilized 2.0 mm to address the patient's concurrent scarring.[16]

We propose that the inferior results on the left side are due to a competing flow phenomenon. The trauma of deep needling triggers an immediate outflow of blood and interstitial fluid. This positive outward pressure gradient likely washes away the topically applied toxin, preventing it from diffusing down to the sebaceous glands effectively.[17] Consequently, the actual amount of toxin reaching the sebaceous glands is likely significantly lower than the injected dose. Much of the solution may remain in the epidermis or be lost to evaporation and surface wiping. This accounts for the slower and less pronounced reduction in pore size (Score 4 at Day 14).

Despite the inferior pore reduction, the microneedling side exhibited distinct advantages in terms of skin texture. In previous reports, this is often attributed to neocollagenesis.[18] However, it is scientifically imperative to correct this timeline. At Day 14, the wound healing cascade is in the proliferative phase, but significant deposition and maturation of Type I collagen takes months. Therefore, the textural smoothness observed on the left side at Day 14 should be attributed to post-traumatic edema and the early accumulation of glycosaminoglycans in the granulation tissue, which temporarily volumizes the skin. While beneficial for the appearance of the patient, clinicians must distinguish this transient swelling from true structural remodeling, which requires longer follow-up to confirm.

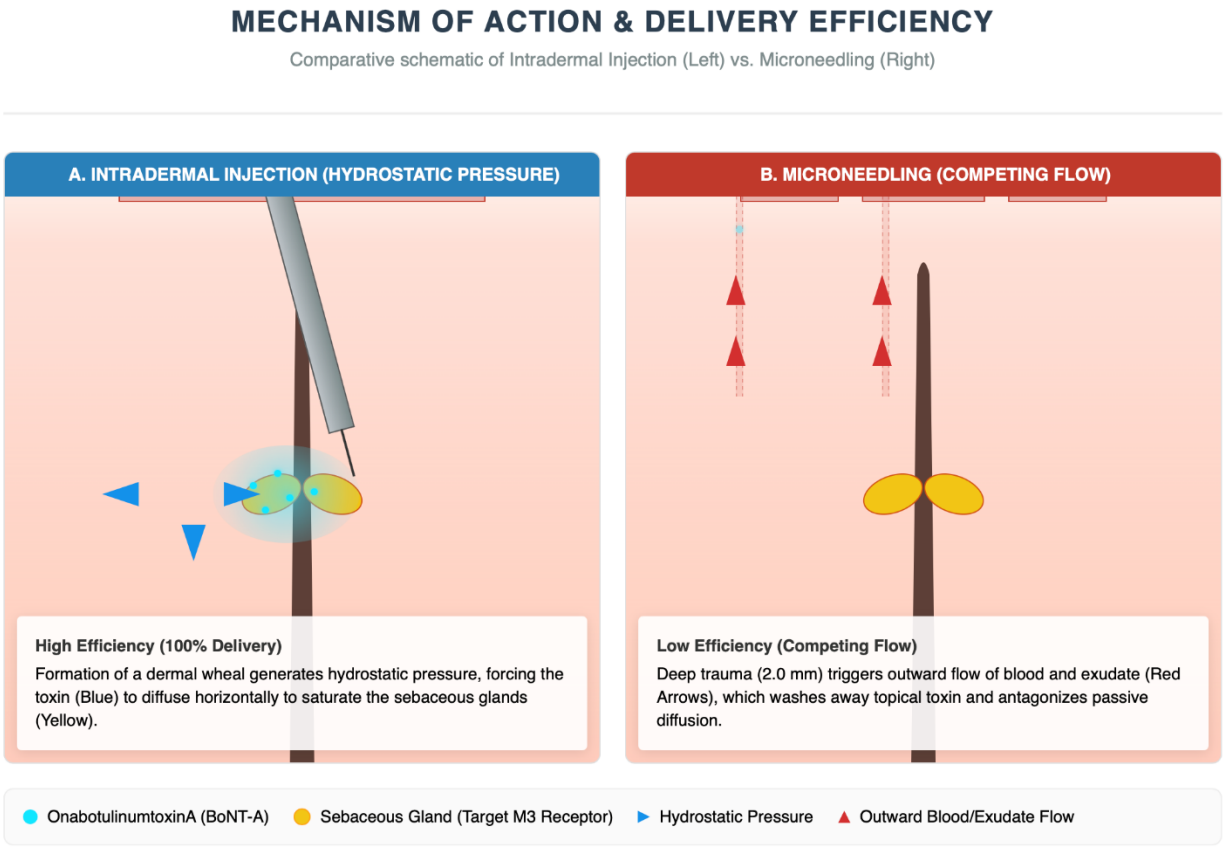

Figure 3. Comparative mechanism of intradermal injection and microneedling.

Both modalities were demonstrated to be safe. The adverse effects were transient and predictable. Intradermal injection caused minimal downtime, limited to hours, whereas microneedling caused inflammation lasting 2 days. Importantly, no frozen face or asymmetry was observed, confirming that the intradermal injection technique, when performed correctly with high dilution, does not affect the deeper muscles of facial expression such as the zygomaticus or risorius.[19]

This study is a single-case report, which limits the generalizability of the findings to the broader population. Additionally, the follow-up period was limited to 14 days. While this is sufficient to observe the onset of BoNT-A action, typically 3–7 days, long-term follow-up of 3–4 months would be required to evaluate the duration of the effect and the long-term impact on collagen remodeling. Future studies should utilize seborrhea-meters and 3D skin analysis systems for more precise quantification.[20]

4. Conclusion

Enlarged facial pores are a multifactorial cosmetic concern requiring targeted therapeutic strategies. This split-face study demonstrates that intradermal injection of Microbotox is the superior modality for achieving a rapid and significant reduction in pore size and sebum production. The direct delivery ensures maximal clinical delivery efficiency of the toxin at the level of the sebaceous glands. Conversely, microneedling-assisted delivery, while less effective for immediate pore contraction, offers synergistic benefits in skin resurfacing and collagen stimulation.

For clinicians, the choice of technique should be guided by the primary pathology of the patient. For patients where seborrhea and pore size are the dominant features, intradermal injection is the gold standard. For patients presenting with a combination of enlarged pores and atrophic scarring, a hybrid approach or the selection of microneedling may be more appropriate, though the limitation of drug

delivery at deeper needle depths due to vascular washout must be considered.

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