PIH: What It Is, Why It Happens, and How We Stop It

Ingredient-level education for understanding any skincare protocol — not just ZO

Staff Training Reference · Dr. V Aesthetics, PLLC · Vaidehi Patel, MD · June 2026

1 WHAT IS PIH — AND WHY DOES IT MATTER IN AESTHETICS

Post-inflammatory hyperpigmentation (PIH) is excess melanin deposited in the skin as a direct response to inflammation or injury. When skin is wounded, irritated, or heated, melanocytes — the pigment-producing cells in the basal epidermis — switch into overdrive. The result: brown, tan, or gray discoloration that typically appears **1–4 weeks after the triggering event**, long after the visible inflammation has resolved.

THE MECHANISM	WHAT TRIGGERS IT IN AESTHETICS
Epidermal PIH (most common)	Melanin deposited in the epidermis. Brown/tan in appearance. Usually resolves with consistent treatment. Results from nearly any controlled-injury treatment — microneedling, RF, chemical peels, laser.
Dermal PIH (deeper, slower)	Melanin drops into the dermis. Gray/blue-brown tone. Slower to resolve — months to years. More common in FP III–VI. Triggered by aggressive treatment or repeated inflammation.
Why aesthetics is high-risk	Every controlled-injury treatment we perform creates the exact inflammation that triggers PIH. Microneedling, RF microneedling, and chemical peels all qualify. The darker the skin type, the more reactive the melanocytes. **Prevention is always easier than treatment.**

2 THE FITZPATRICK SCALE — YOUR PRIMARY RISK GUIDE

The Fitzpatrick scale classifies skin by its melanocyte reactivity and UV response. It is the single most important clinical variable in PIH risk. Higher Fitzpatrick types have more active melanocytes — they respond to inflammation more intensely and for longer.

TYPE	DESCRIPTION	PIH RISK	CLINICAL APPROACH
I	Very fair, always burns, never tans	LOW	Standard protocols. Still prep and protect with SPF — not immune.
II	Fair, burns easily, tans minimally	LOW	Standard protocols. SPF non-negotiable post-treatment.
III	Medium, sometimes burns, tans gradually	MODERATE ■	Pre-treat 4–6 weeks. Conservative settings. Test spot for energy devices. Extended post-care monitoring.
IV	Olive/medium-brown, rarely burns, tans well	HIGH ■■	Mandatory pre-treatment 6+ weeks. Conservative settings always. Test spot required. Avoid IPL.
V	Brown, very rarely burns	VERY HIGH ■■■	Mandatory 6–8 week pre-treatment. Most energy devices require provider sign-off. RF microneedling preferred over IPL/laser.
VI	Deeply pigmented, never burns	VERY HIGH ■■■	Highest risk. Provider assessment required before any energy device. Topical/oral-first approach often safer.

Key rule: When in doubt about Fitzpatrick type, treat as the higher risk category. A FP III patient presenting with melasma history should be managed as FP IV. Melasma, OCP use, or prior PIH history all escalate risk regardless of visual skin tone.

3 THE INGREDIENTS — WHAT THEY DO AND WHY THEY WORK

Understanding the ingredient is more important than knowing the brand. Any product — ZO or otherwise — works because of its active ingredient and its mechanism. Know the mechanism and you can evaluate any product a patient brings in.

INGREDIENT	MECHANISM — WHY IT WORKS	USE CASE	ZO EXAMPLE
Hydroquinone (HQ) 2–4%	Gold standard tyrosinase inhibitor. Directly blocks the enzyme that converts tyrosine to melanin. Most studied depigmenting agent in dermatology. Requires cycling (3 months on, off) to avoid ochronosis.	*Established PIH, melasma. FP III–VI with moderate-severe pigment. Second-line after HQ-free.*	*Pigment Control + Blending Creme (4% HQ, Rx)*
Arbutin / Alpha-arbutin	HQ derivative — slower, gentler tyrosinase inhibitor. Hydrolyzes to HQ in skin but with less irritation. Good for sensitive skin or as a gentler HQ-free alternative.	*FP I–III mild PIH. Maintenance phase. Patients who can't tolerate HQ.*	*Found in Brightalive (arbutin-based brightening complex)*
Kojic acid (1–2%)	Fungal-derived. Chelates copper ions needed for tyrosinase activity, blocking melanin production. Best combined with HQ or glycolic acid — evidence shows combination outperforms either alone.	*Mild-moderate PIH. OTC-accessible. Additive with other brighteners.*	*Found in select ZO brightening blends; also available OTC in many serums*
Azelaic acid (10–20%)	Dicarboxylic acid. Selectively inhibits tyrosinase in hyperactive melanocytes only — does not affect normally pigmented skin. Also anti-inflammatory and antimicrobial. Particularly valuable post-RF where inflammation drives PIH.	*Post-treatment PIH management. Sensitive/reactive skin. Rosacea-prone patients. FP III+.*	*Not a primary ZO ingredient — consider as a standalone adjunct (Rx 20% or OTC 10%)*
Niacinamide (B3, 4–10%)	Inhibits melanosome transfer from melanocyte to keratinocyte — stops pigment from spreading even after it's produced. Also anti-inflammatory, barrier-strengthening, sebum-regulating. Well tolerated by all skin types including sensitive.	*All Fitzpatrick types. Pre- and post-treatment. Barrier support phase.*	*Complexion Renewal Pads contain niacinamide; found in many ZO formulations*
Retinoids (retinol / tretinoin)	Accelerate epidermal cell turnover — pigmented cells shed faster, replacing with unpigmented cells. Tretinoin (Rx) most potent; retinol OTC effective but slower. Also disperses existing melanin granules in keratinocytes. Do not use acutely — introduce wk 3–4 post-treatment.	*All PIH types once skin stable. FP I–IV with intact barrier. Start low and titrate.*	*Retinol Skin Brightener 0.25% / Wrinkle + Texture Repair*

Tranexamic acid (topical / oral)	Blocks plasminogen activator — which triggers keratinocyte-stimulated melanogenesis. Particularly effective for UV-induced and post-procedure pigmentation. Oral TXA (250mg BID) has RCT evidence comparable to 4% HQ for melasma. Topical 2–5% is emerging.	*Moderate-severe PIH FP III+. Oral form when topicals plateau (wk 8–10). FP IV–VI escalation.*	*Not a primary ZO product — prescribe oral TXA separately; recommend topical TXA serums as adjunct*
Vitamin C (L-ascorbic acid 10–20%)	Antioxidant. Inhibits dopaquinone oxidation — an intermediate step in the melanin synthesis pathway. Also neutralizes UV-induced free radicals that trigger melanocyte activation. Unstable — look for stabilized forms (ascorbyl glucoside, AA2G, MAP). Oral 500–1000mg works systemically.	*All skin types. Excellent prevention + treatment support. Layer with SPF in AM.*	*ZO C-Bright 10% Vitamin C Serum (topical); add oral Vit C as adjunct*
SPF 50+ (broad-spectrum)	UV radiation is the #1 activator of melanocyte production. Even minimal UV exposure reverses weeks of brightening. UVA penetrates glass — indoor patients are not safe without SPF. Non-negotiable before, during, and after any PIH treatment or prevention protocol.	*Every patient. Every day. Rain or shine. Non-negotiable.*	*Oclipse Smart Tone SPF 50 (tinted for camouflage while treating)*
Polypodium leucotonos (Heliocare)	Oral antioxidant fern extract. Reduces UV-induced oxidative stress and direct melanocyte stimulation. Clinically shown to reduce photodamage and PIH risk as an adjunct to topical SPF. Very low risk profile. OTC.	*Add-on for sun-reactive patients. FP III+ patients pre- or post-treatment.*	*Not a ZO product — recommend as OTC oral adjunct (Heliocare or equivalent)*

The single-product trap: One tyrosinase inhibitor without turnover support, barrier repair, and UV protection is incomplete. Results plateau and pigment rebounds when stopped. A regimen does four jobs simultaneously — no single product does all four. This is true regardless of brand.

4 PREVENTION — BY FITZPATRICK TYPE

Prevention is where PIH is won or lost. The protocol below applies to any controlled-injury service — microneedling, RF microneedling, or chemical peels. Adjust aggressiveness based on Fitzpatrick type.

	FP I–II (Low Risk)	FP III (Moderate Risk)	FP IV–VI (High Risk)
Pre-treatment prep window	2–4 weeks minimum	**4–6 weeks minimum**	**6–8 weeks; longer if melasma history**
Melanin suppressant	HQ-free preferred (Brightalive, arbutin)	HQ-free first; escalate to 4% HQ if not responding	4% HQ for 6+ weeks pre-treatment; confirm tolerability
Retinoid prep	Retinol 0.25–0.5% PM, 3x/week	Retinol 0.25% — introduce slowly, confirm no irritation	Low-dose retinol only; hold if any irritation present
SPF	SPF 50+ daily — start at booking	SPF 50+ daily — strict. Reapply midday.	SPF 50+ daily — non-negotiable. Treat as a prescription.

Treatment settings	Standard settings per device protocol	Conservative end of recommended range; avoid stacking passes	Most conservative settings; single pass; test spot 48h+ prior
Test spot	Not required; use clinical judgment	Recommended for borderline cases or melasma history	Required. Assess at 48–72h before full treatment.
Post-care monitoring	Standard: 2-week check	48h call/check + 2-week in-person	48h check + 1-week + 4-week in-person. Watch for PIH wk 1–4.

5 TREATMENT — WHEN PIH OCCURS

When PIH occurs despite prevention, the response should be prompt and staged. The earlier intervention begins, the better the outcome — every week of UV exposure without treatment deepens the pigment.

STAGE	TIMEFRAME	INTERVENTION	FITZPATRICK NOTE
1 Stabilize	Wk 1–2	Stop all retinoids, exfoliants, actives. Barrier support only. Strict SPF 50+ twice daily. Begin HQ-free brightener (Brightalive / arbutin-based). Document and photograph.	*All types: same first step. FP IV–VI: ensure SPF is mineral-based (zinc oxide).*
2 Brighten	Wk 3–8	Introduce retinol 0.25% PM 2–3x/week once skin stable. Add niacinamide layer. Add topical Vit C (ZO C-Bright or equivalent) in AM. Oral Vitamin C 500–1000mg/day.	*FP III: add 4% HQ if not responding at wk 6. FP IV–VI: 4% HQ at wk 4 if tolerating.*
3 Escalate (oral)	**Wk 8–12 if plateaued**	Add oral tranexamic acid 250mg BID x 8–12 weeks. Screen for DVT/PE history, pregnancy, renal impairment first. Add Heliocare 240mg daily. Continue all topicals.	*FP III–VI: primary escalation. FP I–II: rarely needed — reassess protocol compliance first.*
4 In-office (if needed)	**Wk 10+ if not responding**	Superficial mandelic or lactic acid peel (skin must be stable). Low-fluence Q-Sw Nd:YAG 1064nm for FP III–IV (test spot required). Avoid IPL in FP IV+. Avoid chemical peels if PIH is still active.	*FP IV–VI: Q-Sw Nd:YAG only. No IPL. No medium/deep peels. Provider-only decision.*

■ NEVER perform chemical peels on active PIH — evidence shows poor/no response in up to 66.7% of cases and risk of worsening. Wait until PIH is visibly improving before any in-office escalation.

6 HOW TO TALK TO PATIENTS — CONVERTING THE REGIMEN

Conversion is not about selling harder — it's about making the science understandable so the patient makes a good choice. Frame around outcome and risk, not products.

PATIENT SAYS	WHAT'S REALLY HAPPENING	YOUR RESPONSE
"Do I really need all of these products?"	Cost concern; doesn't understand the regimen logic	"Each product does a different job — one suppresses the pigment cells, one speeds up how fast your skin renews, and SPF makes sure UV doesn't undo all of it. One product can't do all three."
"I already use X brand — can I just use that?"	Loyal to existing routine; doesn't want to switch	"Let me check what's in it. If it has [ingredient], that covers [step]. We may be able to keep it and just fill the gaps."

"How long until I see results?"	Expectation calibration needed	"Most patients start to see lightening at 4–6 weeks. Melanin clears at the rate your skin renews — we can speed that up but we can't skip the biology. Consistent use is what drives results."
"I don't want to use hydroquinone."	HQ concern — often heard about side effects	"Totally reasonable — we start HQ-free with Brightalive which works the same pathway without HQ. If the response is slower than we want at 6–8 weeks, we can revisit. Most patients do well on the HQ-free path."