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CASE REPORT

Successful Treatment of Cutaneous Foreign Body Granuloma with JAK Inhibitor Abrocitinib and Prednisone: A Case Report

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Background: Foreign body granuloma (FBG) formation is linked to chronic persistent cutaneous inflammation, representing a severe delayed complication characterized histologically by infiltration of multinucleated giant cells and aggregation of lymphocytes. In filler-induced FBG following cosmetic injections, implanted materials represent a key driver of sustained inflammatory responses. Achieving complete resolution remains challenging, with current therapeutic outcomes for FBG being suboptimal. Emerging evidence suggests that Janus kinase (JAK) inhibitors may constitute a promising therapeutic strategy for refractory granulomatous conditions.

Objective: This case report describes the successful management of FBG using JAK inhibitors and synthesizes existing literature to evaluate the efficacy, safety, and potential mechanisms of abrocitinib in treating filler-induced cutaneous FBG.

Methods: We present a case of post-filler FBG that presents with multiple smooth-surfaced, hemispherical lesions (3–5 mm in diameter) distributed across the entire facial region. The patient was treated with oral abrocitinib (100 mg daily) and prednisone (30 mg daily, tapered over 9 weeks). Clinical outcomes were assessed weekly for 13 weeks through serial clinical photography, dermoscopy, reflectance confocal microscopy, and multispectral imaging. Adverse events, including rash exacerbation, vomiting, dizziness, and fever, were systematically monitored. A comprehensive literature review was conducted to elucidate JAK inhibitors' therapeutic rationale in filler-associated FBG.

Results: The patient achieved complete granuloma resolution within 13 weeks following failed corticosteroid monotherapy. No treatment-related adverse effects were observed during the one-month follow-up period, supporting the favorable safety profile of this therapeutic approach.

Conclusion: This report provides preliminary evidence for JAK inhibitors' efficacy in managing refractory filler-induced FBG. Large-scale controlled trials are warranted to validate long-term safety and therapeutic benefits.

Keywords: JAK inhibitors, foreign body granuloma, dermal fillers, cosmetic procedures, abrocitinib

Introduction

Age-related cutaneous changes, including wrinkle formation and facial volume loss, have driven the growing popularity of dermal filler injections as minimally invasive rejuvenation procedures. According to the International Society of Aesthetic Plastic Surgery (ISAPS), global filler procedures increased from 650,000 (2000) to 3.7 million (2018), establishing this as the second most performed aesthetic intervention worldwide.[1,2] Concurrently, complication rates have escalated proportionally, encompassing both immediate (<30 days) and delayed (>30 days) adverse events.[3] Early-phase complications include hematoma, edema, erythema, and hypersensitivity reactions, while delayed manifestations feature foreign body granulomas (FBGs), chronic infections, and material migration.[3]

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FBG represents a severe delayed complication characterized histologically by multinucleated giant cell infiltrates and lymphocyte aggregation. Current epidemiological data indicate an incidence ranging from 0.01% to 14%, influenced by filler composition and injection technique.[4]

Current therapeutic strategies include:[5,6] First, systemic/topical antibiotics (limited evidence). Second, intralesional/oral corticosteroids (variable efficacy, potential relapse). Third, Surgical excision (definitive but scar-forming, particularly problematic in facial regions). These therapeutic regimens demonstrate limited applicability in specific patient subsets, necessitating ongoing exploration of viable alternatives by clinicians.

The Janus kinase (JAK) family - comprising JAK1, JAK2, JAK3, and TYK2 - regulates cytokine signaling through intracellular tyrosine kinase activity.[7] Dysregulation of this pathway underlies numerous immune-mediated conditions, positioning JAK inhibitors as promising therapeutic agents. Recent dermatological applications demonstrate efficacy in alopecia areata, atopic dermatitis, and connective tissue disorders,[8] with emerging evidence supporting their role in granulomatous pathologies. Notably, the expanding utilization of JAK inhibitors in dermatological therapeutics has garnered significant attention, with empirical evidence from multiple investigators substantiating their efficacy in managing FBG.

We present a novel case of refractory facial FBG following collagen-polydeoxyribonucleotide (PDRN) composite filler injection, successfully managed through combined JAK inhibitor (abrocitinib) and corticosteroid therapy. This report integrates a systematic literature review to elucidate mechanistic rationales and clinical evidence supporting JAK inhibition in filler-induced granulomas.

Case Presentation

A 38-year-old Chinese female presented to our hospital with multiple erythematous papules and pruritus on her face persisting for one week. The symptoms developed 24 hours after receiving mesotherapy for facial rejuvenation, which involved manual injection of a collagen and PDRN filler combination. Initial treatment at an external facility included oral prednisolone (15 mg twice daily) for 3 days, intramuscular dexamethasone (5 mg daily) for 3 days, and topical applications of metronidazole gel, mometasone furoate ointment, and hydrocortisone butyrate ointment. While transient improvement occurred, symptoms exacerbated following medication discontinuation, manifesting as elevated, pruritic papules on the forehead and bilateral cheeks. The patient had no underlying diseases, no history of any specific food or drug reactions, and no history of exposure to other toxic substances. Her family history were unremarkable.

Physical examination revealed multiple smooth-surfaced, hemispherical lesions measuring 3–5 mm in diameter across the entire facial region accompanied by localized temperature elevation. (Figure 1A). Multispectral dermatoscopic analysis confirmed inflammatory activity in the treatment area (Figure 1B). Dermatoscopic evaluation demonstrated linear and branching vascular patterns with atypical distribution (Figure 1C). Reflectance confocal microscopy (RCM) identified hyperkeratosis, mild spongiotic changes in the stratum spinosum, and dermal deposits of highly refractive material with dendritic cells (Figure 1D).

Comprehensive laboratory investigations, including complete blood count (CBC), CRP, hepatic/renal function tests, lipid profile, infectious serologies (hepatitis B/C, HIV), tuberculosis screening (T-SPOT), and tumor markers (CA125, CA153, CA199), yielded normal results. Histopathological confirmation was deferred due to patient refusal.

Clinical resolution occurred by week 9 (Figure 1E, I and M). Longitudinal monitoring via serial multispectral imaging (Figure 1F, J and N), Dermatoscopy (Figure 1G, K and O), and RCM (Figure 1H, L and P), demonstrated progressive vascular normalization, reduction of dermal deposits, and decreased inflammatory signals. Therapeutic intervention comprised abrocitinib (100 mg daily) combined with a prednisone taper (30 mg daily, reduced over 9 weeks) (Figure 2). Follow-up assessments at 4 weeks post-discontinuation confirmed sustained remission without recurrence. CBC and blood biochemistry analyses demonstrated no clinically significant abnormalities. No adverse drug reactions were reported throughout the therapeutic course.

The patient provided written informed permission to have any accompanying photos and case details published. The Hospital Ethics Committees of the Fifth People's Hospital of Hainan Province approved to publish the case details.

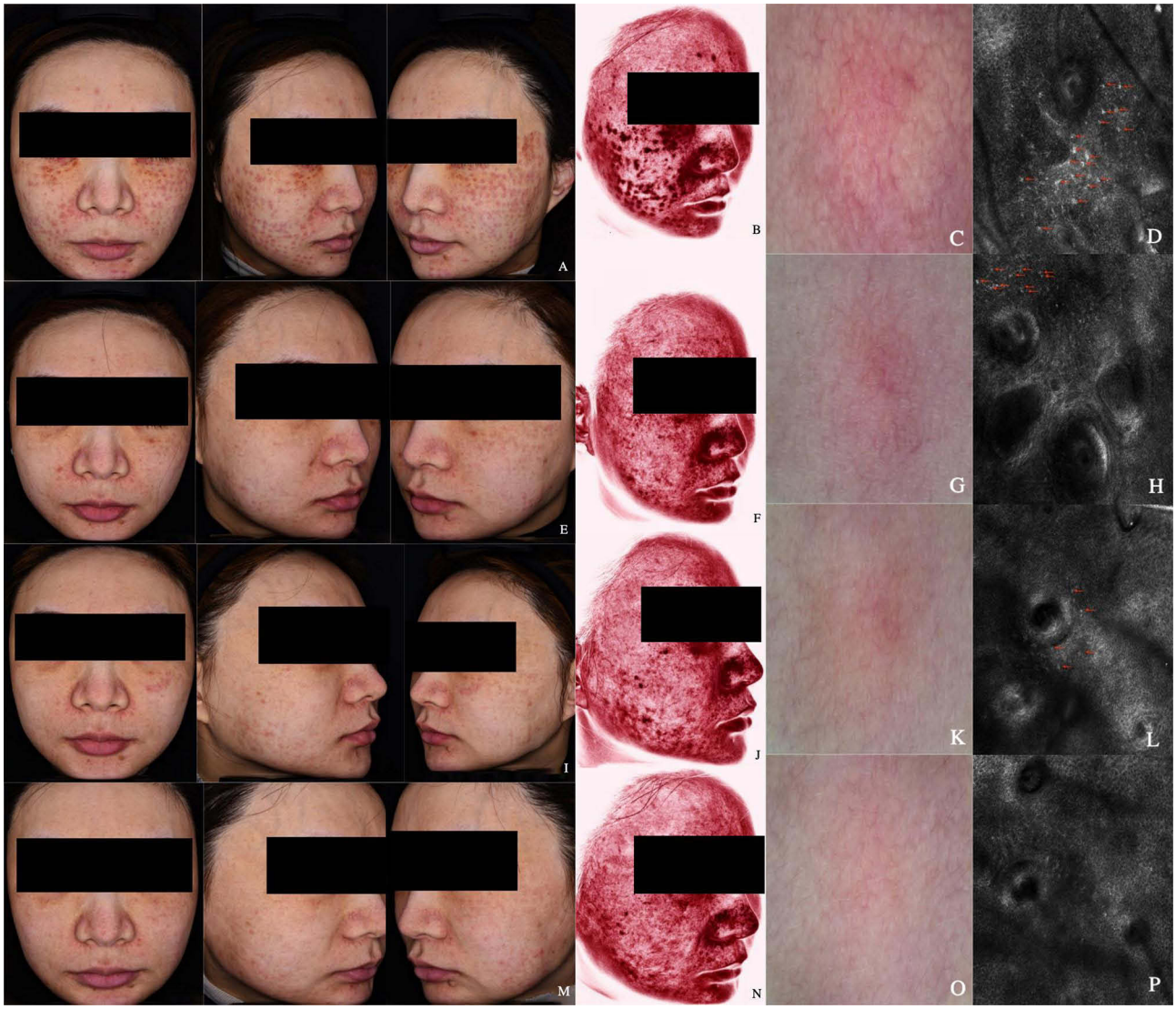

Figure 1 Clinical Photography, Multispectral dermatoscopic analysis, dermatoscopy and RCM of the patient. (**A**) Multiple smooth-surfaced, hemispherical lesions measuring 3–5 mm in diameter were identified across the entire facial region prior to treatment. (**E, I** and **M**) Clinical resolution occurred at 5, 9, and 13 weeks post-treatment. (**B**) Longitudinal monitoring via serial multispectral imaging revealed multiple inflammatory signals across the entire facial region prior to treatment. (**F, J** and **N**) Inflammatory signals showed significant reduction at 5, 9, and 13 weeks post-treatment. (**C**) Dermatoscopic evaluation demonstrated linear and branching vascular patterns with atypical distribution prior to treatment. (**G, K** and **O**) Dermatoscopy demonstrated progressive vascular normalization at 5, 9, and 13 weeks post-treatment. (**D**) RCM identified hyperkeratosis, mild spongiotic changes in the stratum spinosum, and dermal deposits of highly refractive material with dendritic cells prior to treatment. The dermal deposits (consisting of highly refractive material and containing dendritic cells) are indicated by red arrows. (**H, L** and **P**) RCM showed reduction of dermal deposits at 5, 9, and 13 weeks post-treatment.

Discussion

Foreign Body Granulomas (FBGs) are traditionally classified as a Type IV Delayed-Type Hypersensitivity (DTH) reaction in immunology, typically manifesting as late-onset responses. Previous literature indicates that FBGs may develop between 1 week to several years post-injection,[3,9] though rare cases emerge within 24 hours.[10] Granulomatous reactions involve macrophage activation and aggregation. Macrophage-derived IL-12 and IL-18 drive naïve $CD4^+$ T cell differentiation into Th1 cells. Subsequent IFN-γ secretion by Th1 and NK cells regulates DTH expression and macrophage activation, culminating in macrophage accumulation at DTH sites via the $CD4^+$ Th1-cytokine-macrophage axis.[11] Multinucleated Giant Cells (MGCs), formed through macrophage fusion, contribute to FBG cellular composition. STAT6-dependent transcriptional activation mediated by IL-4/IL-13 signaling facilitates MGC formation, while fibrosis in FBGs is linked to TGF-β1, IL-13, IL-4, and CSF-1.[12]

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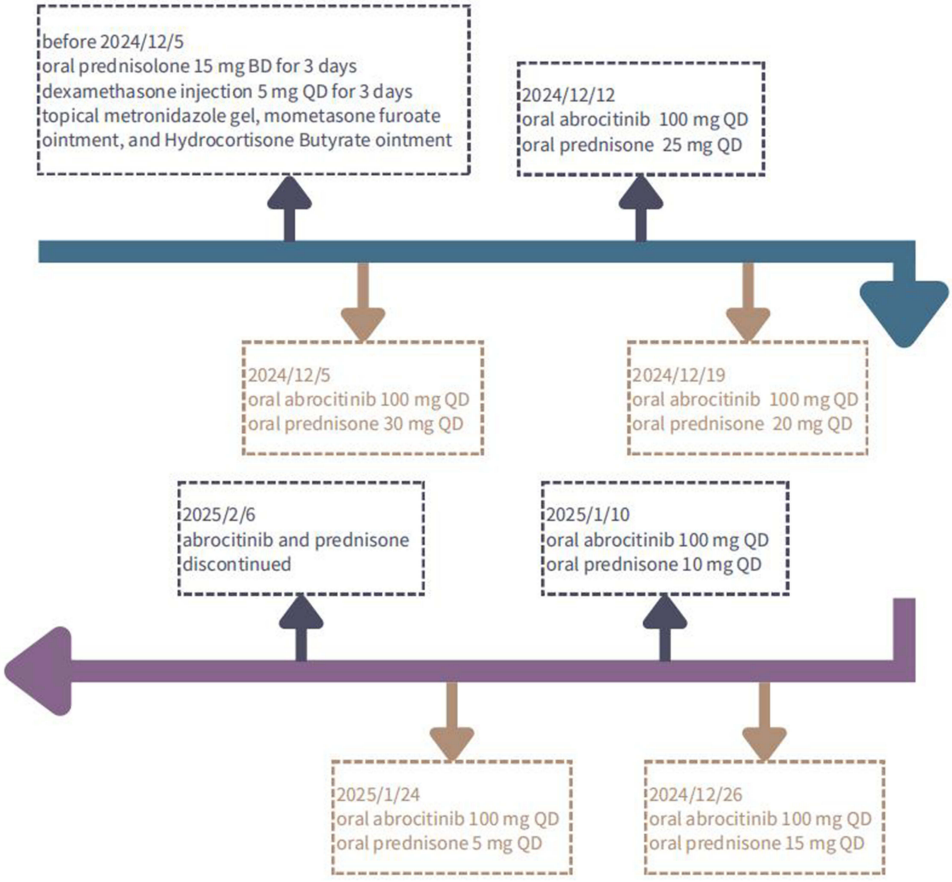

Figure 2 Treatment Timeline. (QD), Once Daily. (BD), Twice Daily.

Histopathological examination remains the gold standard for FBG diagnosis. However, invasive skin biopsies often result in scarring, particularly problematic for facial lesions. Non-invasive imaging tools like RCM circumvent this limitation, with FBGs appearing as hyperreflective circular structures amidst chronic inflammation.[13–15] In this case, a definitive diagnosis of filler-induced FBG was established through clinical history (post-cosmetic injection) and RCM findings.

The treatment of FBG includes glucocorticoids, minocycline, allopurinol, colchicine, cyclosporine, laser therapy, and surgical excision. Among these, systemic glucocorticoids constitute the first-line therapeutic strategy, with oral prednisone administered at an initial dosage of 30 mg daily representing the recommended standard of care.[16] The mechanism of prednisone in treating FBGs may involve suppressing immune responses, inhibiting fibroblast proliferation, and reducing collagen deposition.

Glucocorticoid therapy for FBGs is limited by systemic side effects, including immunosuppression, metabolic disruption, and adrenal suppression.[17] Notably, glucocorticoid insensitivity has been substantiated across multiple clinical cases,[18–23] a phenomenon that underscores the clinical imperative to develop alternative therapeutic strategies for FBG management. In case series reported by other investigators, JAK inhibitors have been implemented in glucocorticoid-insensitive FBG cases, demonstrating promising therapeutic outcomes.

Our patient's FBG persisted despite glucocorticoid treatment but resolved completely following JAK inhibitor administration (abrocitinib), with no adverse effects observed during follow-up.

The possible therapeutic mechanism of JAK inhibitors in FBGs involves multi-level immunomodulation through interconnected biological processes. Firstly, at the cellular regulation level, JAK-STAT pathway inhibition disrupts T cell-macrophage homeostasis by preventing rapid immune activation against pathogens or stimuli,[23] while simultaneously suppressing STAT6-dependent MGC formation that would normally occur through IL-4/IL-13

signaling.[12,24] This dual cellular effect is complemented by molecular targeting mechanisms that work synergistically. Specifically, JAK inhibitors transcriptionally inhibit fibrosis-promoting cytokines including TGF-β1, IL-13, IL-4, and CSF-1,[12,25] while also attenuating macrophage polarization and T cell-mediated activation pathways that would otherwise drive proinflammatory cytokine responses.[26] Furthermore, they achieve downstream effects by downregulating characteristic M1 macrophage markers such as TNFα, CXCL10, and NOS2,[27] which are critical mediators of inflammatory processes. Finally, this therapeutic intervention completes its multimodal action by effectively blocking elevated IFN-γ-driven JAK-STAT signaling pathways that sustain granulomatous inflammation,[28] thereby addressing multiple facets of the disease pathophysiology through coordinated molecular and cellular interventions.

A literature review on JAK inhibitors for the treatment of FBG indicates promising efficacy and a favorable safety profile, despite the limited number of available studies. Epidemiological data reveal FBG predilection for females, with frequent injection sites including the face, buttocks, and extremities. Collagen, hyaluronic acid, (Polymethyl methacrylate) PMMA, and (poly-L-Lactic acid) PLLA are frequently implicated fillers. Diagnostic methods vary, with 60% of reported cases confirmed histologically.[18–23] Therapeutic regimens encompass JAK inhibitor monotherapy (tofacitinib/abrocitinib), combination therapy with glucocorticoids (prednisone/methylprednisolone), or antihistamine (fexofenadine). JAK inhibitors demonstrate consistent efficacy across studies, with no treatment-related adverse events (AEs) reported during follow-up. Complete symptom resolution was achieved within 2–12 months (Table 1).

As JAK inhibitors are metabolized in the liver via the CYP450 system, primarily through the CYP3A4 enzyme, concomitant use of medications that are also metabolized by CYP3A4 (clarithromycin, econazole, itraconazole, and tipranavir) should be avoided during therapy.[29] Moreover, the composition of injectable materials and disease chronicity have been considered as modifiable factors that may significantly influence therapeutic success rates.

Notably, JAK inhibitors are associated with dramatic increase in infection risk, even with fatal outcome, cardiac toxicity, clot formation. Other documented adverse reactions encompass reversible leukopenia, acneiform eruptions, cephalalgia, transaminase alterations, nasopharyngitis, and emesis.[29] Fortunately, in both our reported cases and prior literature, no treatment-related adverse events (AEs) were observed during follow-up.

Table 1 The Treatment of Foreign Body Granulomas with JAK Inhibitors Reported in the Literature

Author	Mansouri[18]	Wang[19]	Ianhez[20]	Yang[21]	Lopez[22]	Li[23]
Nation	Iran	China	Brazil	China	America	China
Case (n)	1	3	1	1	1	1
Gender [male/female, (n)]	0/1	0/3	0/1	1/0	0/1	0/1
Average age (years)	50	37	59	41	55	32
Symptoms	Hyperpigmented lesions, swelling, nodules, and pain	Swelling, nodules	Swelling, nodules, and pain	Papules and nodules	Swelling, nodules, and pruritus	Papules and nodules
Materials	Oils	Collagen or PLLA	PMMA	Ink	Hyaluronic acid	Collagen and hyaluronic acid
Inducing Factors	ND	ND	The COVID-19 vaccination	ND	ND	ND

(*Continued*)

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Table 1 (Continued).

Author	Mansouri[18]	Wang[19]	Ianhez[20]	Yang[21]	Lopez[22]	Li[23]
Location	Buttocks	Face	Face	Shoulders and arms	Face	Face
Diagnostic method	Skin Biopsy	Skin biopsy or MRI	Dermoscopy and skin biopsy	Skin biopsy	Clinical diagnosis	Clinical diagnosis
Treatment	Tofacitinib 5 mg BD	Tofacitinib 5 mg QD OR 5 mg BD	Tofacitinib 5 mg BD	Abrocitinib 100mg QD and prednisone 10–30mg QD	Abrocitinib 100mg QD and fexofenadine 180 mg BD	Abrocitinib 100 mg QD and methylprednisolone 16mg QD
Year of publication	2024	2024	2024	2024	2024	2025
Average onset time	1 year	5.3 months	3 years	13 years	6 weeks	2 months
Follow-up (months)	12	NA	12	6	2	2
Adverse effects	No	No	No	No	ND	No
Recovery rate	100%	100%	100%	100%	100%	100%

Abbreviations: ND Not Described; NA Not Available; PLLA poly-L-Lactic acid; PMMA Polymethyl methacrylate.

This case highlights abrocitinib maybe a promising treatment choice as a safe, effective alternative for glucocorticoid-resistant FBGs. While preliminary clinical outcomes are promising, further mechanistic studies and controlled trials are warranted to optimize JAK inhibitor protocols in granulomatous disorders.

Ethics Statement

The publications of images were included in the patient's consent for publication of the case. The Hospital Ethics Committees of the Fifth People's Hospital of Hainan Province approved to publish the case details.

Consent Statement

Informed consent was provided by the patient for publication of the case.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this work.

References

1. Singh K, Nooreyezdan S. Nonvascular complications of injectable fillers-prevention and management. *Indian J Plast Surg*. 2020;53(3):335–343. doi:10.1055/s-0040-1721872
2. Ahn CS, Rao BK. The life cycles and biological end pathways of dermal fillers. *J Cosmet Dermatol*. 2014;13(3):212–223. doi:10.1111/jocd.12100
3. Hong GW, Hu H, Chang K, et al. Review of the adverse effects associated with dermal filler treatments: part i nodules, granuloma, and migration. *Diagnostics*. 2024;14(15):1640. doi:10.3390/diagnostics14151640
4. Zhang FF, Xu ZX, Chen Y. Delayed foreign body granulomas in the orofacial region after hyaluronic acid injection. *Chin J Dent Res*. 2020;23(4):289–296. doi:10.3290/j.cjdr.b867893
5. Trinh LN, McGuigan KC, Gupta A. Delayed granulomas as a complication secondary to lip augmentation with dermal fillers: a systematic review. *Surg J*. 2022;8(1):e69–e79. doi:10.1055/s-0042-1743524
6. Philipp-Dormston WG, Goodman GJ, De Boulle K, et al. Global approaches to the prevention and management of delayed-onset adverse reactions with hyaluronic acid-based fillers. *Plast Reconstr Surg Glob Open*. 2020;8(4):e2730. doi:10.1097/GOX.0000000000002730
7. Virtanen A, Spinelli FR, Telliez JB, et al. JAK inhibitor selectivity: new opportunities, better drugs? *Nat Rev Rheumatol*. 2024;20(10):649–665. doi:10.1038/s41584-024-01153-1
8. Muddebihal A, Khurana A, Sardana K. JAK inhibitors in dermatology: the road travelled and path ahead, a narrative review. *Expert Rev Clin Pharmacol*. 2023;16(4):279–295. doi:10.1080/17512433.2023.2193682
9. Lucey P, Goldberg DJ. Complications of collagen fillers. *Facial Plast Surg*. 2014;30(6):615–622. doi:10.1055/s-0034-1396904
10. Baranska-Rybak W, Lajo-Plaza JV, Walker L, et al. Late-onset reactions after hyaluronic acid dermal fillers: a consensus recommendation on etiology, prevention and management. *Dermatol Ther*. 2024;14(7):1767–1785. doi:10.1007/s13555-024-01202-3
11. Kobayashi K, Kaneda K, Kasama T. Immunopathogenesis of delayed-type hypersensitivity. *Microsc Res Tech*. 2001;53(4):241–245. doi:10.1002/jemt.1090
12. Pagán AJ, Ramakrishnan L. The Formation and Function of Granulomas. *Annu Rev Immunol*. 2018;36:639–665. doi:10.1146/annurev-immunol-032712-100022
13. Capodiferro S, Sportelli P, Limongelli L, et al. Delayed sclerosing granulomatous reaction to dermal filler injection of poly-hydroxyethyl-methacrylate suspended in hyaluronic acid: histochemical and confocal laser scanning microscopical analysis. *Clin Case Rep*. 2019;7(11):2215–2219. doi:10.1002/ccr3.2478
14. Scivetti M, Lucchese A, Ficarra G, et al. Oral pulse granuloma: histological findings by confocal laser scanning microscopy. *Ultrastruct Pathol*. 2009;33(4):155–159. doi:10.3109/01913120902976562
15. Gao X, Xiang W. Reflectance confocal microscopy as a tool for the diagnosis of labial foreign body granuloma caused by red tattoo pigment: a case report. *Skin Res Technol*. 2023;29(1):e13261. doi:10.1111/srt.13261
16. Lee JM, Kim YJ. Foreign body granulomas after the use of dermal fillers: pathophysiology, clinical appearance, histologic features, and treatment. *Arch Plast Surg*. 2015;42(2):232–239. doi:10.5999/aps.2015.42.2.232
17. Pofi R, Caratti G, Ray DW, et al. Treating the side effects of exogenous glucocorticoids; can we separate the good from the bad? *Endocr Rev*. 2023;44(6):975–1011. doi:10.1210/endrev/bnad016
18. Mansouri P, Farshi S. The new frontier of jak inhibitors: significant therapeutic response to tofacitinib in a patient with granulomatous reaction to filler in the buttocks. *J Cosmet Dermatol*. 2024;23(12):3924–3926. doi:10.1111/jocd.16578
19. Wang J, Chen Z, Wu L, et al. Tofacitinib for managing granuloma formation after dermal filler injection: three case reports and literature review. *J DermatolTreat*. 2024;35(1):2338281. doi:10.1080/09546634.2024.2338281
20. Ianhez M, Miot HA, Caetano LV, et al. Sarcoidosis and polymethyl methacrylate (PMMA) granulomas following COVID-19 vaccination (ChAdOx1): successful treatment with tofacitinib. *Indian J Dermatol*. 2024;69(4):328–332. doi:10.4103/ijd.ijd_56_24
21. Yang Y, Yan J, Yang J, et al. Janus kinase inhibitor abrocitinib as an off-label treatment for tattoo granuloma with uveitis (TAGU). *Australas J Dermatol*. 2024;65(3):297–299. doi:10.1111/ajd.14248
22. Lopez MHP, Guenin SH, Laborada J, et al. Post-hyaluronic acid filler reaction treated with abrocitinib: a case report. *J Drugs Dermatol*. 2024;23(1):1355–1356. doi:10.36849/JDD.7271
23. Li Z, Lu J, Wu M, Yu B, Yang Z. Successful treatment of delayed onset nodules after dermal fillers injection with abrocitinib: a case report. *Clin Cosmet Invest Dermatol*. 2025;18:955–959. doi:10.2147/CCID.S524045
24. Fortelny N, Farlik M, Fife V, et al. JAK-STAT signaling maintains homeostasis in T cells and macrophages. *Nat Immunol*. 2024;25(5):847–859. doi:10.1038/s41590-024-01804-1
25. Stark GR, Darnell JE Jr. The JAK-STAT pathway at twenty. *Immunity*. 2012;36(4):503–514. doi:10.1016/j.immuni.2012.03.013
26. Nyirenda MH, Nijjar JS, Frleta-Gilchrist M, et al. JAK inhibitors disrupt T cell-induced proinflammatory macrophage activation. *RMD Open*. 2023;9(1):e002671. doi:10.1136/rmdopen-2022-002671
27. Lescoat A, Lelong M, Jeljeli M, et al. Combined anti-fibrotic and anti-inflammatory properties of JAK-inhibitors on macrophages in vitro and in vivo: perspectives for scleroderma-associated interstitial lung disease. *Biochem Pharmacol*. 2020;178:114103. doi:10.1016/j.bcp.2020.114103
28. Wang A, Singh K, Ibrahim W, et al. The promise of JAK inhibitors for treatment of sarcoidosis and other inflammatory disorders with macrophage activation: a review of the literature. *Yale J Biol Med*. 2020;93(1):187–195.
29. Miot HA, Criado PR, de Castro CCS, Ianhez M, Talhari C, Ramos PM. JAK-STAT pathway inhibitors in dermatology. *An Bras Dermatol*. 2023;98(5):656–677. doi:10.1016/j.abd.2023.03.001