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# JANUS KINASE INHIBITORS AS A BREAKTHROUGH TREATMENT FOR REFRACTORY DISSECTING CELLULITIS OF THE SCALP

Ali Al-Mamoori[1], Sajjad Ghanim Al-Badri[2], Sabry Babiker H. Sayed[3], Wael Al-Daraji[4]

[1] Department of Dermatology, Baghdad Teaching Hospital, Baghdad, Iraq
[2] College of Medicine, University of Warith Al-Anbiyaa, Karbala, Iraq
[3] Faculty of Medicine and Health Sciences, International University of Africa, Khartoum, Sudan
[4] Dermatology Department, Ain Shams University Hospital, Cairo, Egypt

**Corresponding author's e-mail:** Sabry.babiker@gmail.com

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## ABSTRACT

*Background:* Dissecting cellulitis of the scalp (DCS) is a rare, chronic neutrophilic dermatosis that is often refractory to conventional therapies.

*Case report:* We present a 29-year-old male with treatment-resistant DCS who achieved rapid and sustained remission following off-label use of tofacitinib, a Janus kinase (JAK) inhibitor. Previous therapies, including antibiotics, corticosteroids, and isotretinoin, had failed. After 9 weeks of tofacitinib 10 mg daily, the patient showed marked improvement and maintained remission for 6 months without side effects.

*Conclusion:* This case supports JAK inhibitors as a promising therapeutic strategy in refractory DCS, highlighting their potential role in managing challenging inflammatory dermatoses.

## KEYWORDS

Dissecting cellulitis of the scalp, JAK inhibitors, tofacitinib, neutrophilic dermatosis, follicular occlusion tetrad

## LEARNING POINTS

- *Novel therapeutic breakthrough:* This is one of the first documented cases demonstrating successful monotherapy with tofacitinib in refractory dissecting cellulitis of the scalp (DCS), achieving rapid and sustained remission where all standard treatments had failed.
- *Translational value for internists:* Given the overlap between DCS and other autoimmune/inflammatory disorders commonly encountered by internists (e.g., rheumatoid arthritis, ulcerative colitis), this case supports considering Janus kinase (JAK) inhibitors for complex inflammatory dermatoses beyond current indications.
- *Clinical impact in practice:* The case highlights the practical, off-label use of JAK inhibitors in dermatology, providing internists and multidisciplinary teams with a viable option for patients suffering from severe, treatment-resistant conditions.

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## INTRODUCTION

Dissecting cellulitis of the scalp (DCS), also known as perifolliculitis capitis abscedens et suffodiens or Hoffman disease, is a chronic inflammatory disorder of the scalp characterized by boggy, suppurative nodules that are often associated with patchy hair loss[1]. Follicular occlusion is considered a key pathogenic mechanism in DCS, and the condition often presents alongside other follicular occlusive disorders such as acne conglobate, hidradenitis suppurativa (HS), and pilonidal cysts. These conditions collectively comprise the follicular occlusion tetrad, which is unified by a shared pathogenesis involving follicular hyperkeratosis, follicular rupture, and secondary neutrophilic inflammation[2]. Despite the use of systemic antibiotics, corticosteroids, and isotretinoin, many patients with DCS have incomplete responses or suffer frequent relapses, making long-term management challenging[3].

Transcriptomic studies in HS have identified numerous differentially expressed genes (DEGs), many of which are influenced by Janus kinase (JAK) activity and downstream signal transducer and activator of transcription (STAT) signalling. Because the JAK–STAT pathways regulate the expression of multiple pro-inflammatory cytokines involved in HS, therapeutic inhibition of this signalling axis has emerged as a rational strategy for disease modulation[4-6].

Tofacitinib, a pan-JAK inhibitor primarily targeting JAK1 and JAK3, has demonstrated clinical benefit in patients with recalcitrant HS, further supporting the potential relevance of JAK–STAT pathway inhibition in neutrophilic dermatoses[7].

Similarly, a recent case report described a patient with long-standing, treatment-resistant DCS who achieved rapid and sustained remission after initiating upadacitinib, a selective JAK1 inhibitor, with no reported adverse events[8].Given these findings and the overlapping pathophysiological features between HS and DCS, we present a case of severe, treatment-resistant DCS in a young male patient who showed marked improvement following off-label treatment with tofacitinib.

## CASE DESCRIPTION

A 29-year-old Albanian male presented with a 5-year history of painful, draining nodules and patches of scarring alopecia on the scalp, consistent with DCS. The patient reported persistent symptoms despite multiple standard treatments over several years.

His past medical history was notable only for chronic smoking, averaging 40 cigarettes daily. He denied any similar lesions on other parts of his body, including the axillae and groin.

Previous management included topical clindamycin and clobetasol propionate 0.05% lotion, alongside three to four sessions of intralesional corticosteroid injections (triamcinolone 5 mg/ml). Systemic therapies comprised oral isotretinoin 40 mg daily for 12 months, doxycycline 100 mg twice daily for 14 months, and two separate 3-month courses of rifampicin 300 mg twice daily combined with clindamycin 300 mg twice daily. Despite adherence, these treatments resulted in only partial or transient improvements.

On physical examination, the occipital scalp demonstrated multiple coalescing, fluctuant nodules with sinus tracts and extensive areas of cicatricial alopecia *(Fig. 1)*.

Routine laboratory investigations—including complete blood count, thyroid-stimulating hormone, vitamin D, ferritin, glycated haemoglobin (HbA1c), liver function tests, lipid profile, and blood pressure—were within normal limits. A bacterial culture obtained prior to initiation of systemic immunosuppression yielded no growth.

After thorough discussion regarding off-label treatment and obtaining informed consent, the patient was started on tofacitinib 10 mg once daily. At the 9-week follow-up, he reported a marked reduction in scalp inflammation and pain. Clinical examination revealed significant reduction in pustules, shrinkage of sinus tracts, and absence of visible drainage *(Fig. 2)*. By this time, he achieved clinical remission of active inflammatory lesions.

The patient has maintained stable remission for 6 months, with no reported adverse effects or abnormal laboratory findings during treatment.

## DISCUSSION

DCS is a chronic, relapsing neutrophilic dermatosis that presents significant therapeutic challenges, particularly in

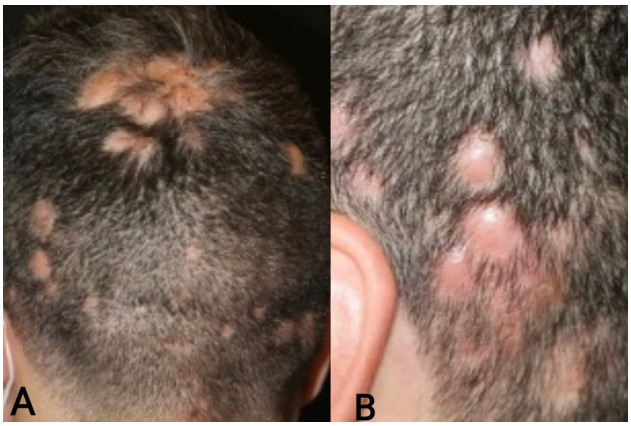



*Figure 1. Pre-treatment occipital scalp showing multiple coalescing nodules, sinus tracts, and extensive cicatricial alopecia.*

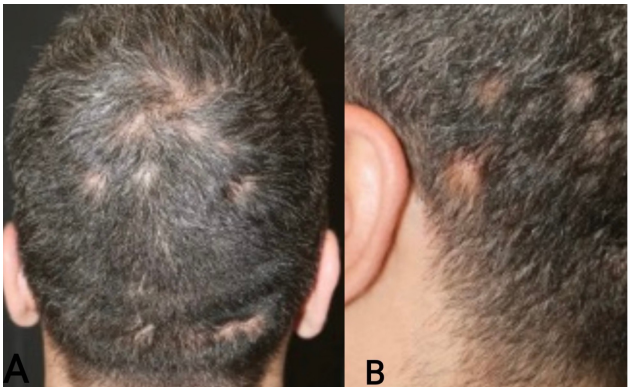



*Figure 2. Post-treatment image after 9 weeks of tofacitinib therapy, showing significant reduction in nodules and resolution of inflammation.*

cases unresponsive to conventional treatments. It shares pathogenic features with HS, acne conglobata, and pilonidal disease—collectively forming the follicular occlusion tetrad—characterized by follicular hyperkeratosis, rupture, and secondary neutrophilic inflammation[2].

Given this overlap, many of the novel therapies for DCS are based on therapeutic experience in HS. Agents targeting tumour necrosis factor (TNF)-α, interleukin (IL)-17, IL-36, IL-1, and small-molecule Janus kinase inhibitors have shown promise in HS and are now being explored in DCS[5,6]. The JAK–STAT signalling pathway has been increasingly recognized as a critical mediator of immune dysregulation in HS, prompting interest in JAK inhibition as a therapeutic approach for neutrophilic dermatoses, including DCS[4,5].

Tofacitinib, a pan-JAK inhibitor primarily targeting JAK1 and JAK3, has demonstrated clinical benefit in patients with recalcitrant HS[7]. It is Food and Drug Administration-approved for several autoimmune and inflammatory disorders, including rheumatoid arthritis, psoriatic arthritis, ulcerative colitis, polyarticular juvenile idiopathic arthritis, and ankylosing spondylitis. However, its use in dermatologic diseases like DCS remains off-label[8,9]. The growing body of evidence supporting its efficacy in inflammatory skin disorders underscores its potential for broader therapeutic application.

Support for JAK inhibitors in DCS is increasing, with one report of sustained remission in a patient treated with upadacitinib, a selective JAK1 inhibitor. In another case successful management of DCS with a combination of ixekizumab (IL-17A inhibitor) and tofacitinib was reported, suggesting a synergistic role between biologics and JAK inhibitors in severe disease[7,10].

Our case adds to this emerging evidence by demonstrating successful monotherapy with tofacitinib in a young male patient with refractory DCS. Remarkably, the patient achieved clinical remission within 9 weeks, with sustained control over 6 months and no reported side effects. This is notable, especially compared to earlier reports that required combination regimens for disease control.

## CONCLUSION

JAK inhibitors demonstrate significant potential for patients with DCS who have failed other treatment options. These findings highlight the importance of exploring targeted therapies for complex and treatment-resistant conditions. Here, we report the second case in the literature. Further studies are essential to optimize dosing, identify biomarkers, and understand long-term efficacy and safety. JAK inhibitors provide a renewed sense of hope for those in whom all other options have failed, paving the way for improved outcomes.

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