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JAK inhibitors as salvage therapy in refractory hidradenitis suppurativa: a real-world case series

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Introduction
Hidradenitis suppurativa (HS) is a chronic, debilitating inflammatory dermatosis with substantial unmet therapeutic needs. Despite approved biologics, a subset of patients with moderate-to-severe HS experience inadequate response or secondary loss of efficacy. Janus kinase inhibitors (JAKi) provide multi-cytokine pathway modulation through the JAK–STAT axis implicated in HS inflammation, yet real-world evidence across different JAKi remains limited. We report clinical outcomes of three JAKi used as salvage therapy in refractory HS and contextualize our findings with the available literature.

Materials and Methods
We retrospectively reviewed 7 patients with moderate-to-severe HS refractory to conventional therapies who received oral JAKi: tofacitinib, upadacitinib, or ivarmacitinib. Disease severity and patient-reported impact were assessed during routine follow-up using established clinical measures, including IHS4 and Hurley staging. Patients were monitored monthly for at least 1 month, with subsequent follow-up visits based on clinical course. Clinical response was evaluated by assessing improvements in inflammatory lesions, drainage/abscess activity, and pain.

Results
Five of seven patients (71.4%) achieved an initial clinical response, with marked improvement in pain and purulent discharge typically within the first month. JAK1-selective agents (upadacitinib and ivarmacitinib) demonstrated rapid improvement in key symptoms in most treated cases. A dose-dependent response was observed with upadacitinib: the 30 mg dose provided better control of deep-seated inflammation compared to the 15 mg dose, which was associated with residual drainage in one patient. Tofacitinib (JAK1/JAK3) showed heterogeneous outcomes, inducing remission in one patient but failing to adequately control disease in others. Rapid recurrence was observed following treatment interruption, suggesting a suppressive rather than curative effect. No severe adverse events were reported during the observed follow-up period.

Conclusions
In this real-world case series, JAK inhibitors—particularly JAK1-selective agents—showed promise as a salvage option for refractory moderate-to-severe HS, with rapid improvement in key symptoms. Responses were heterogeneous, and higher dosing may be required for deep or fibrotic disease components, while interruption may lead to quick relapse. Larger controlled studies are needed to define optimal dosing strategies, durability, and the patient subgroups most likely to benefit.