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Review Paper

Icotrokinra in Plaque Psoriasis: A New Oral Treatment Approach

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ABSTRACT

Plaque psoriasis is a chronic, immune-mediated inflammatory skin disorder that significantly affects patients' quality of life. Beyond physical symptoms, the condition is often associated with psychological distress and comorbidities. Although a wide range of treatment options are available including topical therapies, phototherapy, systemic agents, and biologics, each has certain limitations related to efficacy, safety, cost, or convenience of administration. Biologic therapies targeting key inflammatory pathways, particularly interleukin (IL)-17 and IL-23, have demonstrated high efficacy in achieving skin clearance. However, their requirement for parenteral administration can reduce patient adherence and long-term acceptability. The currently available oral therapies provide lower levels of skin clearance compared to biologics. Icotrokinra is a novel oral peptide that selectively inhibits the IL-23 receptor, a critical mediator in the pathogenesis of psoriasis .Clinical evidence from Phase 2 and Phase 3 trials has demonstrated that icotrokinra produces rapid and significant improvements in disease severity, with a substantial proportion of patients achieving PASI 75, PASI 90, and even complete skin clearance with high efficacy and safety. icotrokinra represents a promising advancement in the management of moderate-to-severe plaque psoriasis. By combining biologic-level efficacy with the convenience of oral dosing, it has the potential to address current treatment gaps and improve patient adherence and outcomes

INTRODUCTION

Plaque psoriasis, commonly referred to as psoriasis vulgaris, is a chronic inflammatory skin disease caused by the immune system. It typically presents well–demarcated, erythematous plaque with a layer of silvery-white scales, often making the skin feel dry and irritated [1,2]. This is the most frequently seen form of psoriasis, accounting for the majority of cases. The condition usually persists throughout life, with periods where symptoms worsen followed by times of improvement. Flare-ups can be brought on by triggers such as infections, stress, certain

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medications, or even minor skin injuries. While some people experience only mild symptoms, others may have more severe and widespread involvement that can affect their comfort and confidence [1,2].

These patches often develop on the elbows, knees, scalp, back, and lower spine, though they can also involve the nails and skin folds. In some cases, the condition may extend beyond the skin and affect the joints, leading to psoriatic arthritis. It has also been linked to other health issues, including heart disease, risk of infection, metabolic problems, and mental health concerns like depression. Early diagnosis and proper management are important to control symptoms, improve quality of life, and reduce the risk of long-term complications [1,2]

Psoriasis can be thought of as the result of an interaction between genetic tendency and external triggers, leading to its onset and periodic flare-ups. Genetics play an important role in this disease; certain genes, particularly those linked to the HLA-Cw6 type, are associated with an earlier onset and more severe forms of psoriasis [1,2].

Various environmental and lifestyle factors can trigger or worsen psoriasis in susceptible individuals. Common triggers include infections such as streptococcal throat infections, skin injuries (known as the Koebner phenomenon), emotional stress, and certain medications like beta-blockers, lithium, and antimalarials. Sudden stopping of corticosteroids can also lead to flare-ups [2,3]. In addition, habits like smoking, alcohol use, and conditions such as obesity and metabolic syndrome may further aggravate the disease [1,2].

About 29.5 million adults are affected by psoriasis all over the world [2,4]. It is considered as adult disease as its prevalence is more when compared to children. Both male and female are affected by psoriasis, with early onset in females with genetic predisposition. The bimodal age of onset with the first peak at 30-39 years and second peak at 60-69 years in males and decade earlier in females [1,4].

Treatment for plaque psoriasis depends on the severity of the disease. For mild cases (BSA<5%) topical therapies such as corticosteroids, vitamin D analogs, coal tar, retinoids, salicylic acid and anthralin are used. Phototherapy is used when inadequate response is observed while using topical therapies [1,2].

For moderate to severe disease (BSA>10%) systemic non biologic therapy is recommended which includes methotrexate, cyclosporine, acitretin [1,2].

Biologics are highly effective for moderate to severe psoriasis. Biologic therapy includes TNF - α inhibitors, IL-17 inhibitors, IL-12/23 inhibitors, and IL-23 inhibitors [1,2]. Biologics are monoclonal antibodies targeting inflammatory mediators, offering sustainable improvement and improved quality of life [1,2, 5]. IL-17 and IL-23 help in achieving fast, high level clear skin with high efficacy and safety profile, but biologic therapies require parenteral administration which is not convenient for long term use [2,5,6].

Oral systemic medicines like apremilast, a phosphodiesterase-4 inhibitor and deucravacitinib, a selective tyrosine kinase 2 inhibitor, have made psoriasis treatment easier and more convenient, since they can be taken as tablets and are usually well tolerated. However, they don't clear the skin as effectively as biologic treatments. Because of this, there is still a need for oral options that are both easy to use and as effective as the latest biologic therapies [5,6,12].

Icotrokinra (JNJ-77242113)is a newer oral medication that works by specifically blocking the IL-23 receptor, an important pathway involved in psoriasis. It has been studied in several Phase 2 and Phase 3 clinical trials, where it has shown promising results[6,7]. Research suggests that icotrokinra may provide effectiveness similar to IL-23 biologic treatments, in oral dosage form which reduces the inconvenience of patients [6,8].

In this review, we discuss findings from clinical

studies that evaluate the effectiveness and safety of icotrokinra as an oral IL-23 receptor inhibitor for moderate-to-severe plaque psoriasis [5,6].

MECHANISM OF ACTION

Icotrokinra is an oral IL-23 receptor inhibitor. It is a macrocyclic peptide. With sub-10- pM affinity icotrokinra binds to IL-23R and silencing the downstream cytokine response which includes IL-17A, IL-17F, IL-22, and interferon (IFN)-γ by inhibiting the activation of JAK2/TYK2-STAT3 signaling cascade. It does not affect IL-12 dependent STAT4 activation which is confirmed by human immune cell assay [6,9,10,11

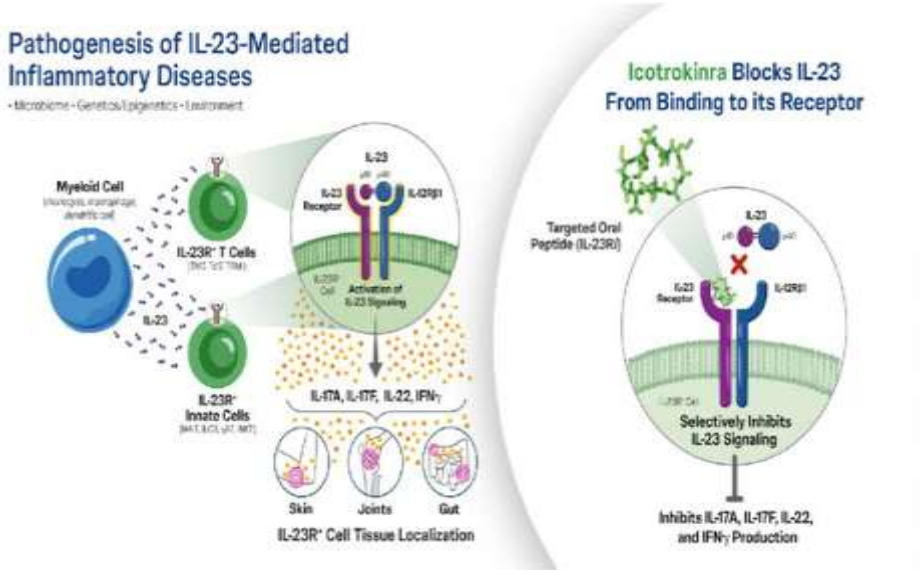

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Fig:1 pathogenesis of IL-23 mediated plaque psoriasis and mechanism of action of icotrokinra [7]

Icotrokinra pharmacokinetic data is from Preclinical, Phase 1 and Phase 2 studies. They showed consistent, dose-dependent suppression of IL-23 driven cytokine release in human blood. It also demonstrated strong anti-inflammatory effects in experimental models of skin inflammation caused by IL-23, suggesting its potential to effectively control psoriasis-related inflammation [6,9]. Its half-life is 9-12 hours; steady state is attained within 7 days and stored at room temperature. In Phase 3 studies, icotrokinra is given 200mg once daily or 100mg twice daily orally. The drug showed high gastrointestinal stability and linear pharmacokinetics after oral administration without absorption enhancers. Protein binding is 50% and excreted in feces. Risk of drug –drug interactions are low because it is not metabolized by cytochrome P450 enzyme [6,9-11].

Icotrokinra is administered 95 healthy subjects in a randomized, double blinded, placebo-controlled Phase 1 study.it showed fast absorption, in 2-5 hours peak plasma concentration [9,10]. Icotrokinra is not suggested in the pregnant women because the safety and efficacy data is lacking, as it is understudied in this population. Icotrokinra is not used in patients with known hypersensitivity to the drug or its excipients [6,11]. In phase 3 studies drug showed safety and efficacy in adult patients and pediatrics ≥12 years weighing above 40kgs[6,11].

SAFETY AND EFFICACY DATA OF ICOTROKINRA FROM CLINICAL TRIALS

Many ongoing clinical trials are conducting icotrokinra for treating multiple immune-mediated inflammatory diseases, mainly for treating psoriasis. In plaque psoriasis Phase 2 and Phase 3 clinical trials are done which showed high efficacy

and safety profile than other oral drugs like deucravacitinib [6,8,9,13,]

Two phase 2 and four Phase 3 trials are conducted for fixing dose and evaluating safety and efficacy of the oral icotrokinra.

Table :1 summary of clinical trial done on the icotrokinra

CLINICAL TRIAL and N	FRONTIER 1 N=255	FRONTIER 2 N=227	ICONIC ADVANCE 1 N=774	ICONIC ADVANCE 2 N=731	ICONIC LEAD N=684	ICONIC TOTAL N=311
DESIGN	Phase 2, double blind,randomized, placebo dose-finding, controlled trail	52 week Extension of frontier 1	Phase 3,placebo, controlled, and active-randomized,double blind, comparator-controlled	Phase 3,placebo,controlled, and active-randomized, double blind, comparator-controlled	Phase3, multicenter, randomized, double-blind, placebo, controlled	Phase 3,multicenter, randomized, doubleblind, placebo,controlled, parallelgrp. interventionaI trial
POPULATION	Adults (≥ 18 years of age) had moderate-to-severe plaque psoriasis	Same as frontier 1	Adults (>18yrs of age) had moderate-to-severe plaque psoriasis	Same as iconic advance 1	Adults and adolescents (>12yrs) with moderate-to-severe,plaque psoriasis	Adults(> 18 year) and adolescents (>12 to<18 years) with plaque psoriasis
DOSING ARM	ICO 25mg OD (n = 43) ICO 25mg BD (n = 41) ICO 50mg OD (n = 43) ICO 100mg OD (n = 43) ICO 100mg BD (n = 42) PBO (n = 43)	Patients who received ICO continued the same dose as frontier 1 PBO → Icotrokinra 100 mg once	ICO (n = 311) DCR (n = 307) PBO (n = 156) subjects assigned to PBO or DCR switched to ICO at W16 or W24, respectively	ICO (n = 322) DCR (n = 327) PBO (n = 82)	ICO (n=456) PBO (n=228)	ICO 200 mg OD(n=208) PBO(n=103)
END POINT	PASI 75 at 16 weeks	PASI 75 at 52 weeks	PASI 90 at 16 weeks & IGA score of 0/1 in the ICO group	PASI 90 at 16 weeks & IGA score of 0/1 in the ICO group	IGA 0/1 and PASI 90 at 16 week	IGA 0/1, hf-PGA 0/1,

			vs. PBO group IGA 0/1, IGA 0, PASI 75, PASI 90, PASI 100 in ICO vs. DCR group at 16 and 24 weeks	vs. PBO group IGA 0/1, IGA 0, PASI 75, PASI 90, PASI 100 in ICO vs. DCR group at 16 and 24 weeks		ss-IGA at 16 weeks.
EFFICACY	**PASI 75 at week 16** 37% (ICO 25 mg QD) 51% (ICO 25 mg BID) 58% (ICO 50 mg QD) 65% (ICO 100 mg QD) 79% (ICO 100 mg BID) 9% (PBO group) p<0.001	**PASI 75 at week 52** 49% (ICO 25 mg QD) 58% (ICO 25 mg BID) 70% (ICO 50 mg QD) 65% (ICO 100 mg QD) 76% (ICO 100 mg BID) 66% (PBO +100 mg group) p < 0.001	**IGA 0/1**: ICO vs.PBO: 68% vs.11% ICO vs DCR: 68% vs 50% **PASI 90 at 16 weeks**: ICO vs PBO: 55% vs. 4% ICO vs DCR: 55% vs 30% All p<0.001	**IGA 0/1:** ICO vs. PBO: 70% vs. 9% ICO vs. DCR: 70% vs. 54% **PASI 90 at 16 weeks**: ICO vs PBO: 57% vs 1% ICO vs. DCR: 57% vs 34% All p<0.001	**IGA 0/1:** ICO vs. PBO: 65% vs 8% **PASI 90at 16 weeks:** ICO vs. PBO: 50% vs 4%	**IGA 0/1:** ICO vs. PBO: 57% vs. 6% **hf-PGA 0/1:** ICO vs. PBO: 42% vs. 26% **ss-IGA 0/1** ICO vs. PBO: 66% vs.11%
C0MMON ADVERSE EVENT	Frontier 1-ICO vs PBO Diarrhea: 5% vs. 2% Nervous system disorders: 5% vs. 2% covid 19-11% vs 12% Nasopharyngitis - 5% vs 7% Frontier 2 - ICO nasopharyngitis –18%, URTI-10%, COVID-19 .5%		ICO vs PBO vs DCR Nasopharyngitis -6% vs. 5% vs. 9% URIs -4% vs. 3% vs. 5% GI- 7% vs. 6% vs. 10%		ICO vs PBO Nasopharyngitis and URI -7% vs 7% GI - 6%vs6%	ICO vs PBO Nasopharyngitis-12%vs 11% URI -4% vs 5%
REFERENCE	6,8,9	6,8,9,13	6,9,14	6,9,14	6,11,17	6,15,18

The frontier 1 trial along with frontier 2 which 52 weeks extension of frontier 1 determined the optimal oral dose of icotrokinra along with its safety and efficacy in adult plaque psoriasis patients. Frontier 1 is Phase 2, dose-finding, double-blind, randomized, placebo-controlled trial using PASI 75 as end point in 16 weeks period evaluated the effectiveness of 5 different doses of icotrokinra in total 255 subjects.[6,8,9,13]

The five dosing regimens are (25 mg once daily, 25 mg twice daily, 50 mg once daily, 100 mg once daily, or 100 mg twice daily; n = 212) or placebo (n = 43) for 16 weeks. Improvement in participant condition is observed at 4th week and at 16 weeks the responses are 37% with 25 mg OD, 79% with

100 mg BID, compared with 9% in the placebo group reached PASI 75.[6,8,9,13]

In the frontier 2 trial, the participants who took icotrokinra in frontier 1 trial received the same dose till 52 weeks and icotrokinra 100mg BID is given to participants who took placebo at 16th week. Higher level responses like PASI 90 and PASI 100 are observed in 60% and 40% of participants with 100mg BID respectively.[6,8,9,13]

In the FRONTIER-1 trial, adverse events occurred in icotrokinra vs placebo groups were 44–62% vs 51% on placebo. The most common issues were mild upper respiratory infections like nasopharyngitis (5% vs 7%), diarrhea (5% vs. 2%), headache, and cough.in nervous system rates were 5% vs. 2%. Serious events were rare (COVID-19, infected cyst, and a suicide attempt), and none were related to the treatment. There were no deaths, cancers, or major cardiovascular problems, and no clear dose-related safety concerns.[8,9]

In FRONTIER 2 trial,59% of the participants experienced at least one AE, most common were nasopharyngitis, upper respiratory infection, and COVID-19 and their rates were 18%, 10% and 5% respectively.GI AE were 11% at week 16 and 6% at week 52. SAEs were seen in 4% of participants which were deemed unrelated to the treatment. [9,11]

ICONIC ADVANCE 1 and ADVANCE 2 were Phase 3 Phase multicenter, randomized, double-blind, placebo- and comparator-controlled, clinical trials that assessed the efficacy and safety profile of icotrokinra versus both deucravacitinib (oral TYK2 inhibitor), and placebo, in adults with plaque psoriasis. Participants were randomized in 2:1:2 ratio to administer icotrokinra 200 mg once daily (n=633), placebo (n=238), or deucravacitinib 6 mg once daily (n=634). Investigator's Global Assessment (IGA) score of 0/1, with at least a 2-grade improvement, and achievement of ≥90% improvement in PASI (PASI 90) at week 16 is the primary endpoint and comparing it with placebo , deucravacitinib at 16 and 24 weeks PASI75, PASI90, PASI100, IGA 0/1, and IGA 0.[6,9,14,15]

By week 16 the Icotrokinra successfully met both primary endpoints at high efficacy although clinical improvement was observed at week 4 for PASI75 and by week 8 for PASI90. In ADVANCE 1 trial, at week 16 participants receiving icotrokinra achieved higher response rates than those receiving placebo, with IGA 0/1 responses of 68% vs. 11% and PASI 90 responses of 55% vs. 4%. In ADVANCE 2 trial, the corresponding rates are 70% vs. 9% and 57% vs. 1%. (all $p<0.001$ for both trials). [6,9,10,16]

At week 16, icotrokinra achieved secondary end points with higher efficacy than deucravacitinib. In ADVANCE 1, PASI75, PASI90, and IGA 0/1 were achieved in 74% vs. 57%, 55% vs. 30%, and 68% vs. 50%, respectively. In ADVANCE 2, the corresponding rates were 77% vs. 61%, 57% vs. 34%, and 70% vs. 54%, respectively (all $p<0.0001$ for both trials).Also in ADVANCE 1, PASI 100 was 31% vs. 11% and IGA 0 was 37% vs. 16%; and in ADVANCE 2, PASI 100 was 32% vs. 14% and IGA 0 was 37% vs. 17% (all $p<0.001$). [6,14,16]

At week 24 further improvements are seen, in ADVANCE 1, PASI75, PASI90, and PASI100 were achieved in 82% vs. 64%, 66% vs. 41%, and 41% vs. 16%, respectively; IGA 0/1 and IGA 0 occurred in 74% vs. 52% and 48% vs. 21%, respectively (all $p<0.0001$). In ADVANCE 2, corresponding rates were 83% vs. 66%, 65% vs. 43%, and 33% vs. 16% for PASI75, PASI90, and PASI100, and 68% vs. 55% and 40% vs. 21% for IGA 0/1 and IGA 0, respectively (all $p\leq0.0002$). [6,14,16]

Patient-reported outcomes favored icotrokinra over deucravacitinib. On the Psoriasis Symptoms and Signs Diary (PSSD), 24% of patients receiving

icotrokinra versus 9% of those receiving deucravacitinib achieved a symptom score of 0 at week 16. [6,14,16]

In both ADVANCE 1 and ADVANCE 2, nasopharyngitis and upper respiratory tract infections were the most reported adverse events. Up to 24 weeks, the rates of adverse events in the icotrokinra versus deucravacitinib groups were 6% vs. 9% for nasopharyngitis and 4% vs. 5% for upper respiratory tract infections. [6,9,10,]

ICONIC-LEAD is a phase 3, randomized, double-blind, placebo-controlled trial evaluating adults and adolescents (≥12 years) with moderate-to-severe plaque psoriasis(n=684) [11,15,19]. characterized by body surface area (BSA) ≥10%, PASI ≥12, and an Investigator's Global Assessment (IGA) score ≥3. For 16 weeks participants were given icotrokinra or placebo at 2:1 ratio randomly; At week 24, adult participants who reached PASI 75 or an Investigator's Global Assessment (IGA) score of 0/1 were reassigned in a 1:1 ratio to either remain on icotrokinra or switch to placebo for a 28-week withdrawal period. Treatment could be restarted if at least 50% of the PASI improvement achieved at week 24 was lost. Meanwhile, adolescent participants continued receiving active therapy through week 52. [9,11,15,17]

By week16, greater proportion of participants treated with icotrokinra achieved IGA 0/1 (65% vs. 8%) and PASI 90 (50% vs. 4%) compared with placebo (both p<0.001). Additionally, icotrokinra demonstrated superior rates of complete skin clearance, with more participants attaining IGA 0 (33% vs. 1%) and PASI 100 (27% vs. <1%) versus placebo (both p<0.001). [9,11,15]

Separation from placebo occurred early, with PASI 75 responses observed at week 4 (15% vs. 2%; p=0.002) and PASI 90 responses at week 8 (21% vs. 1%; p<0.001) In participants with baseline scalp involvement (scalp IGA ≥2), ss-IGA 0/1 responses at week 16 were observed in 72% of those treated with icotrokinra versus 15% of those receiving placebo (p<0.001), these improvements were maintained through week 24 among participants who continued icotrokinra (80%). At week 16 those who switched from placebo to icotrokinra showed comparable scalp response rates (77%).[15,17]

Efficacy further increased through week 24, with 74% of participants achieving IGA 0/1, 65% achieving PASI90, 46% reaching complete skin clearance (IGA 0), and 40% achieving PASI100. Improvements in scalp psoriasis were maintained and increased, with 80% of participants on continuous icotrokinra achieving an ss-IGA 0/1 response by week 24. Participants switching from placebo to icotrokinra at week 16 showed comparable scalp outcomes, with a scalp IGA 0/1 rate of 77%. [9,17]

In adolescents (n = 66), icotrokinra showed strong efficacy in week 16. Compared with placebo, a greater proportion of icotrokinra-treated participants achieved IGA 0/1 (84% vs. 27%) and PASI 90 (70% vs. 14%) (both p<0.001). Complete skin clearance was also more frequent with icotrokinra, with higher rates of IGA 0 (41% vs. 5%; p<0.01) and PASI 100 (30% vs. 5%; p<0.05). By week 24, responses further increased, with 86% for IGA 0/1, 89% for PASI 90, and complete clearance observed in 75% for IGA 0 and 64% for PASI 100. [9,17]

At week 24 among adults who were re-randomized (in total 341 responders; 169 continued icotrokinra and 172 switched to placebo) A higher proportion of participants who continued icotrokinra maintained PASI 75 (89% vs. 30%) and PASI 90 (84% vs. 21%) compared with those switched to placebo (both p<0.001).[17,18] .With ongoing therapy, patients did not reach the median time to loss of PASI75 or PASI90 responses, whereas those who discontinued treatment experienced loss of PASI75 at 16.9 weeks and PASI90 at 10.1 weeks.

Similarly, maintenance of clear or almost clear skin (IGA 0/1) was significantly higher with continued treatment (82%) compared to withdrawal (23%; $p < 0.001$).[11,17]

In ICONIC LEAD, icotrokinra demonstrated a safety profile comparable to placebo through week 16 in adults, with similar overall adverse event rates (49% in both groups) and low rates of serious adverse events (1% vs. 3%). Infections, including nasopharyngitis and upper respiratory tract infections (7% in both), were the most common events, and serious infections were rare (< 1%). Gastrointestinal events occurred at similar frequencies (6%), and no deaths were reported. In adolescents, the safety profile was consistent with adults, with lower overall adverse event rates in the icotrokinra group compared with placebo (50% vs. 73%) and in infections (32%vs. 27%). Serious adverse events were infrequent (transient hospitalization for joint pain and pancreatitis secondary to choledocholithiasis) were considered unrelated to treatment, and resolved without sequelae, with no cases of tuberculosis, malignancy, or deaths observed. [15]

The Phase 3 trial ICONIC TOTAL is randomized, double-blind, placebo-controlled study evaluated icotrokinra versus placebo for scalp, and genitals, palms and sores psoriasis with BSA as low as 1% in 311 adolescents and adults (≥ 12 years).Participants were randomized to icotrokinra (n=208) or placebo (n=103) with placebo switch to icotrokinra at week16[20]. Among participants with palmoplantar psoriasis, greater proportions of those treated with icotrokinra achieved clear or almost clear skin on the hands and feet (hf-PGA 0/1: 42% vs. 26%) compared with placebo. Similarly, improvements in other difficult-to-treat sites favored icotrokinra, including scalp psoriasis (ss-IGA 0/1: 66% vs. 11%) and genital psoriasis (sPGA-G 0/1: 77% vs. 21%) (all $p<0.001$), demonstrating consistent efficacy across multiple localized disease areas. [9,18]

At week 52, response rates were 67% for overall IGA 0/1, 72% for scalp ss-IGA 0/1, 85% for genital sPGA-G 0/1, and 62% for hand/foot hf-PGA 0/1. Week 52 response rates were compared with participants who switched from placebo to icotrokinra at week 16. Complete clearance was achieved in a higher proportion of icotrokinra-treated patients, with 44% in IGA 0, 57% in scalp ss-IGA 0, 73%i in sPGA-G 0, and 58% in hf-PGA 0. [9,18]

In ICONIC-TOTAL, safety profiles were comparable between icotrokinra and placebo. Overall adverse events (AEs) occurred in 50% of participants in the icotrokinra group versus 42% in the placebo group, with nasopharyngitis and mild gastrointestinal symptoms being the most common. Serious adverse events were rare (0.5% vs 1.9%) and GI adverse event rates were 7.2% vs. 7.8%. Infection rates were 28.4% vs 21.4%, with serious infections reported in 0% vs 1.0% of participants. One case of melanoma occurred in the icotrokinra group in a patient with a prior history of melanoma. No emergency safety signals or deaths were reported. [9,18]

DISCUSSION

As a first-in-class oral peptide inhibitor of IL-23R, icotrokinra offers a promising treatment option for moderate-to-severe plaque psoriasis. Currently, the US FDA-approved targeted oral therapies are deucravacitinib, a selective TYK2 inhibitor, and apremilast, a PDE4 inhibitor. These agents act on specific intracellular pathways and differ from broad-acting oral immunosuppressants used for psoriasis management. While these therapies are effective for psoriasis, they have limitations in efficacy and potential tolerability issues that can restrict their clinical use. Biologics are highly effective for treating moderate to severe psoriasis, but maintaining long-term injectable therapy is not convenient for most patients. So, the need for oral medication is needed.

Although deucravacitinib outperforms traditional nonbiologic therapies, it still showed lower skin clearance rates than newer IL-23 inhibitors. Icotrokinra is an oral IL-23R antagonist that provides biologic-level efficacy with the convenience of oral dosing. Clinical studies comparing deucravacitinib with icotrokinra have shown that icotrokinra delivered superior efficacy versus deucravacitinib in patients with moderate-to-severe plaque psoriasis. In addition, PASI 100 rates and overall skin clearance IGA 0 outcomes for icotrokinra were observed in clinical trials.

CONCLUSION

The treatment options for moderate-to-severe plaque psoriasis have expanded over time and now include topical therapies, systemic immunosuppressants, biologic agents, and newer targeted oral medications. Biologic treatments are highly effective and can achieve significant skin clearance, but their use may be limited by the need for injections. Oral therapies such as apremilast and deucravacitinib provide a more convenient alternative, although they generally do not match the effectiveness of biologics.

Recent Phase 2 and 3 studies suggest that icotrokinra may help bridge this gap. As a targeted oral peptide, it combines the convenience of a once-daily tablet with promising effectiveness and a favorable safety profile, making it a potential new option for patients seeking both efficacy and ease of use.

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