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Efficacy of Tofacitinib in Atopic Dermatitis such as Vitiligo, Atopic Dermatitis, Chronic Dermatitis, Prurigo Nodularis Lichen Planus and Prologis Simplex

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Abstract **Original Research Article**

***Background*:** Fifteen years have gone by since the approval of a topical treatment for atopic dermatitis (AD) with a novel mechanism of action, despite unmet demand. The effectiveness of topical Janus kinase (JAK) inhibitor therapy in AD patients is uncertain. ***Objectives*:** This study was done for assessment of efficacy of tofacitinib in atopic dermatitis such as vitiligo, atopic dermatitis, chronic dermatitis, prurigo nodularis lichen planus and prologis simplex. ***Methods*:** The cross-sectional Observational study was conducted in the Department of skin and VD, Ashiyan Medical College and Hospital from June 2022 to May 2023. A total of 82 patients of both sexes were included in the study. Data were collected over a period of 12 months and analyzed by appropriate computer based programmed software Statistical Package for the Social Sciences (SPSS), version 24. ***Results*:** In this study, about 31 (37.8%) patients lies between 31 years to 35 years and 22 (26.8%) patients lies between 36 years to 40 years. Mean ± SD of the patients age was 31.5 ± 7.3 years. More than half of the patients 48 (58.50%) were female and 34 (41.50%) patients were male. Most of the patients 52 (63.4%) BMI were in between normal range, 13 (15.9%) were underweight and 17 (20.7%) were overweight. Mean ± SD of the patients BMI was 29.3 ± 5.4 kg/m2. About 36 (43.9%) patients experienced severe itching, 35 (42.7%) patients experienced moderate itching and 11 (13.4%) patients experienced mild itching. Age of onset of the disease of most of the patients 48 (58.5%) were 10 – 12 years, 24 (29.3%) were 13 – 15 years and 10 (12.2%) were 16 – 18 years. Mean ± SD of the patients age of onset of disease was 12.0 ± 9.4 years. ***Conclusion*:** The oral Janus kinase inhibitor tofacitinib citrate may be beneficial in the treatment of moderate to severe AD.

Keywords: Tofacitinib, Atopic Dermatitis, Vitiligo, Atopic Dermatitis, Chronic Dermatitis, Prurigo Nodularis Lichen Planus, Prologis Simplex.

INTRODUCTION

With incidence rates of up to 20% and rising, atopic dermatitis (AD) is a widespread inflammatory skin disorder that affects both adults and children globally. Usually, onset takes place in childhood [1]. Clinical indicators, the distribution and shape of skin lesions, and past characteristics are used to make the diagnosis [2]. High socioeconomic hardship is linked to AD, which affects patient health-related quality of life (HRQoL) and healthcare resource utilization [1, 3, 4]. One of the main symptoms of AD is pruritus, which has a detrimental impact on a patient's HRQoL, especially their mental and sleep quality [4, 5].

The cornerstone of AD treatment consists on topical medications such as calcineurin inhibitors (CNIs), corticosteroids, and emollients [6, 7]. Additional therapies consist of systemic immunosuppressants, topical and oral antibiotics, phototherapy, and refined coal tar [6–8]. The ineffectiveness of nonsteroidal topical treatments, limitations on application to specific body parts, "steroid and CNI phobia," and application site reactions are some of the drawbacks of the available treatments [7, 9]. Systemic adverse effects, skin atrophy (for striae and other atrophic alterations) from topical corticosteroids, and an elevated risk of infections from CNIs are long-term safety issues [6–8]. It has been 15 years since the approval of a new AD medication with a

unique mechanism of action, underscoring the need for additional potent drugs to treat AD despite the persistent unmet medical need [7, 8, 10].

Since AD is a multifaceted disease resulting from a complex interaction between genetic, environmental, and immunological variables, its pathophysiology and aetiology remain unclear [11, 12]. Specifically, the pathophysiology of AD has been linked to T-helper cell (Th)2 cytokines interleukin (IL)-4, IL-5, IL-13, and IL-31 [11, 12]. Recent early-phase clinical trials of dupilumab, an experimental injectable monoclonal antibody that blocks IL-4/IL-13 signaling, have shown notable benefits in persons with moderate-to-severe AD [13, 14].

Many cytokines and growth factors use the Janus kinase (JAK)–signal transducer and activator of transcription (STAT) pathway to transduce signals [15]. One small-molecule JAK inhibitor is tofacitinib [16]. Tofacitinib has been demonstrated to directly block cytokines like IL-4 and cause keratinocytes' JAK–STAT signaling to rapidly attenuate [17]. Patients with mild-to-moderate chronic plaque psoriasis responded well to tofacitinib ointment in phase II research [18], and patients with moderate-to-severe chronic plaque psoriasis responded well to oral tofacitinib in phase IIb and phase III trials [19–22].

METHODOLOGY

The cross-sectional Observational study was conducted in the Department of skin and VD, Ashiyan Medical College and Hospital from June 2022 to May 2023. A total of 82 patients of both sexes were included in the study. Purposive sampling was done according to the availability of the patients who fulfilled the selection criteria. Face to face interview was done to collect data with a semi-structured questionnaire. After collection, the data were checked and cleaned, followed by editing, compiling, coding, and categorizing according to the objectives and variables to detect errors and to maintain consistency, relevancy and quality control. Statistical evaluation of the results used to be obtained via the use of a window-based computer software program devised with Statistical Packages for Social Sciences (SPSS-24).

RESULT

Table I shows that, about 31 (37.8%) patients lies between 31 years to 35 years and 22 (26.8%) patients lies between 36 years to 40 years. Mean ± SD of the patients age was 31.5 ± 7.3 years.

Table I: Distribution of the patients according to age (n = 82)

Age group (in years)	Frequency	%
21 – 25	8	9.8
26 – 30	16	19.5
31 - 35	31	37.8
36 - 40	22	26.8
41 - 45	5	6.1
Total	**82**	**100.0**
Mean ± SD: 31.5 ± 7.3 years		

Figure I shows that, more than half of the patients 48 (58.50%) were female and 34 (41.50%) patients were male.

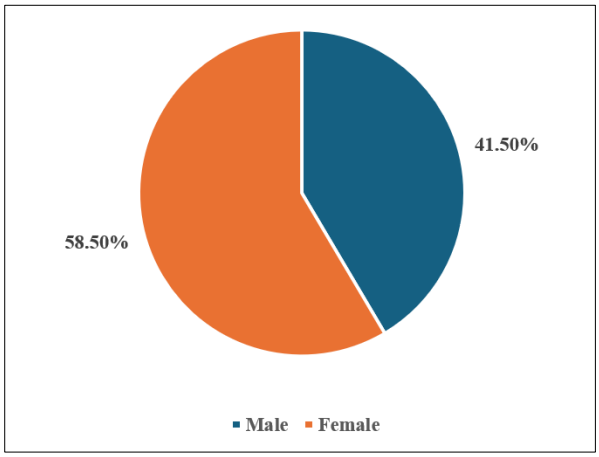

Figure I: Distribution of the patients according to sex (n=82)

Table II shows that, most of the patients 52 (63.4%) BMI were in between normal range, 13 (15.9%) were underweight and 17 (20.7%) were overweight. Mean ± SD of the patients BMI was 29.3 ± 5.4 kg/m^2

Table II: Distribution of the patients according to Body mass index, kg/m2 (n = 82)

Body mass index	Frequency	%
Underweight (18.5 – 24.9)	13	15.9
Normal (25 – 29.9)	52	63.4
Overweight (more than 30)	17	20.7
Total	**82**	**100.0**
Mean ± SD: 29.3 ± 5.4 kg/m^2		

Table III Shows that, about 36 (43.9%) patients experienced severe itching, 35 (42.7%) patients experienced moderate itching and 11 (13.4%) patients experienced mild itching.

Table III: Distribution of the patients according to itching Severity (n=82)

Itching severity	Frequency	%
Mild	11	13.4
Moderate	35	42.7
Severe	36	43.9
Total	**82**	**100.0**

Table IV shows that, age of onset of the disease of most of the patients 48 (58.5%) were 10 – 12 years, 24 (29.3%) were 13 – 15 years and 10 (12.2%) were 16 – 18 years. Mean ± SD of the patients age of onset of disease was 12.0 ± 9.4 years.

Table IV: Distribution of the patients according to age of onset (n = 82)

Age of onset (in years)	Frequency	%
10 - 12	48	58.5
13 - 15	24	29.3
16 - 18	10	12.2
Total	**82**	**100.0**
Mean ± SD: 12.0± 9.4 years		

DISCUSSION

Autoimmune inflammatory dermatosis known as atopic dermatitis (AD) is widespread, recurrent, and systemic. Xeroxis, eczematous lesions, noticeable dryness and redness, and intense, ongoing pruritus are the hallmarks of AD. AD is linked to a high burden, which includes poor quality of life, anxiety, sadness, and sleep disturbance. Emollients, topical corticosteroids, and calcineurin inhibitors are used to treat atopic dermatitis. Immunomodulatory drugs are used to treat refractory illness, albeit they are frequently insufficient for moderate to severe cases. New therapeutic methods have been made possible by advances in our understanding of the pathophysiology of AD.

The cross-sectional Observational study was conducted in the Department of skin and VD, Ashiyan Medical College and Hospital from June 2022 to May 2023. A total of 82 patients of both sexes were included in the study.

In this study, about 31 (37.8%) patients lies between 31 years to 35 years and 22 (26.8%) patients lies between 36 years to 40 years. Mean ± SD of the patients age was 31.5 ± 7.3 years. More than half of the patients 48 (58.50%) were female and 34 (41.50%) patients were male. Most of the patients 52 (63.4%) BMI were in between normal range, 13 (15.9%) were underweight and 17 (20.7%) were overweight. Mean ± SD of the patients BMI was 29.3 ± 5.4 kg/m2. About 36 (43.9%) patients experienced severe itching, 35 (42.7%) patients experienced moderate itching and 11 (13.4%) patients experienced mild itching. Age of onset of the disease of most of the patients 48 (58.5%) were 10 – 12 years, 24 (29.3%) were 13 – 15 years and 10 (12.2%) were 16 – 18 years. Mean ± SD of the patients age of onset of disease was 12.0 ± 9.4 years.

JAK inhibition has been demonstrated in another recent clinical investigation to alleviate AD symptoms. Tofacitinib functionally selectively inhibits signaling by heterodimeric receptors linked to JAK3 and/or JAK1 as opposed to receptors that signal through JAK2 pairs [16, 25]. Signaling via a number of cytokine receptors, such as IL-2, IL-4, IL-7, IL-9, IL-13, IL-15, and IL-21, is blocked by this inhibition [16, 25, 26]. The JAK–STAT system is crucial for Th2 cell development in AD, a Th2-dominant inflammatory skin disease; specifically, JAK1, JAK3, and STAT6 are involved in IL-4 signaling [10–12]. Furthermore, a number of cytokines released by JAK-STAT and Th2 cell proliferation play a crucial role in the inflammatory reactions in AD. By upregulating chemokines and proinflammatory proteins and downregulating antimicrobial peptides and components involved in skin

barrier function, IL-4 has been linked to the deregulation of keratinocyte function in AD [10–12].

Pruritus is also influenced by IL-31 overexpression. By blocking signaling through IL-31, tofacitinib suppression of JAK1/JAK2 may reduce pruritus [27–29]. This may help to explain why the current study's ISI score gains started so quickly. Tofacitinib's likely mode of action in AD is hence suppression of the JAK–STAT pathway and cytokines such IL-4, IL-13, and IL-31 [16, 25, 26]. When combined, the current study's findings, the report proving the effectiveness of the IL-4/IL-13 inhibitor dupilumab [13], and an analysis of targeted biological therapies10 support the idea that the JAK–STAT pathway plays a role in the pathophysiology of AD11 and also support the idea that cytokine inhibition and the JAK–STAT pathway could be used as treatment targets for AD.

In persons with mild-to-moderate AD as defined by PGA, the current trial demonstrated that JAK kinase inhibition with 2% tofacitinib ointment twice daily produced significant improvements compared to vehicle across all effectiveness end points over 4 weeks, including the major end goal. Tofacitinib significantly improved end points compared to the vehicle starting in week 1, suggesting a quick start to efficacy and continued improvement through week 4. Additionally, as early as one day after starting tofacitinib treatment, a significant and statistically significant decrease in pruritus from baseline was noted. This is especially crucial because AD is referred to as the "itch that rashes," and pruritus has a detrimental effect on quality of life [5, 30, 31]. Although the target rate of 3 mg cm^2 was exceeded by the mean ointment application rate per dosage over the treatment period (~2~5 mg cm~2), the observed mean rate was comparable to the rate recorded in patients who were told to apply a thin layer of a topical medicament [32].

Measurable systemic levels of tofacitinib indicated that it had penetrated the skin. The commencement of efficacy with the restoration of normal skin barrier function may be the cause of lower concentrations at week four. The observed trend of greater plasma tofacitinib concentrations with higher treated % BSA is not surprising at week 2, when skin barrier function is probably still compromised. However, this pattern was no longer noticeable by week 4, when there was a noticeable improvement in the disease. Lower concentrations at week four might possibly be explained by decreased patient adherence to treatment of the baseline diseased BSA for the entire four weeks; however, this is not supported by the mean application rate, which remained constant for both treatment groups between days 1 and 2 and for the interval from week 2 to week 4.

Application site reactions, eczema herpeticum incidence, potential elevated risk of infections and cancers, and corticosteroid side effects such atrophy, striae, and suppression of the hypothalamic-pituitary-adrenal axis are among the safety concerns with many of the existing AD treatments. Both tofacitinib and vehicle experienced [6–8]. TEAEs infrequently; however, vehicle experienced more TEAEs and treatment-area TEAEs than tofacitinib. Unlike two individuals receiving vehicle treatment, no tofacitinib-treated patients permanently left the study due to adverse events. There were no reported Serious AEs. After four weeks of treatment, tofacitinib ointment was well tolerated and showed an adequate safety and local tolerability profile in this phase IIa research.

The current study's short duration, lack of an active comparator arm, and variations in equipment and demographics make it difficult to compare it with other AD therapies that are now available or under research. The use of oral tofacitinib in six individuals with refractory moderate-to-severe AD was recently reported to ameliorate AD symptoms as assessed by BSA and the ScoRing of AD (SCORAD) index. Nevertheless, the trial was constrained by its small sample size and lack of blinding or placebo control [33].

CONCLUSION

While JAK inhibitors are still not approved for treating atopic dermatitis, oral tofacitinib has shown promise in treating AD. Levy *et al.*, (2015) demonstrated the effectiveness of oral tofacitinib in a group of cases involving atopic dermatitis [34]. Then, in a clinical trial conducted in 2016, Bissonette *et al.*, showed promising outcomes with topical tofacitinib [35]. More recently, patients' EASI scores improved as a result of clinical trials with the novel JAK ½ inhibitor baricitinib and the JAK 1 inhibitors upadacitinib and abrocitinib [36–39] The encouraging outcomes in cases of severe AD that is refractory imply that oral tofacitinib may be a viable treatment option. Since AD is a complex illness, using novel therapeutic medications is still an interesting prospect.

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