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Original Article

Efficiency of Baricitinib versus Tofacitinib in the Treatment of Vitiligo

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ABSTRACT

Background: *An acquired, idiopathic autoimmune condition called vitiligo is typified by patches of skin, hair, or both that are depigmented. Despite being regarded as a benign condition that mostly affects appearance, vitiligo typically has a significant negative influence on a patient's quality of life and self-esteem and may even put them at higher risk for skin cancer and sunburn.* ***Objectives:*** *The purpose of this study was to compare the effectiveness of baricitinib and tofacitinib in the treatment of vitiligo.* ***Methods & Materials:*** *The Ashiyan Medical College and Hospital's Department of Skin and VD hosted the cross-sectional observational study from June 2022 to May 2023. The study included 40 patients in total, both male and female. Over the course of a year, data was gathered and analyzed using the Statistical Package for the Social Sciences (SPSS), version 24, a computer program.* ***Results:*** *In this study, the mean age of the patients was 32.2 ± 1.3 years for the Baricitinib group and 38.6 ± 2.3 years for the Tofacitinib group. In the Baricitinib group, 11 patients (55.0%) were male, while in the Tofacitinib group, 13 patients (65.0%) were female. The mean duration of the disease was 6.2 ± 1.3 years for the Baricitinib group and 9.6 ± 2.3 years for the Tofacitinib group. Additionally, the mean treatment duration was 2.2 ± 1.3 months for the Baricitinib group and 3.6 ± 2.1 months for the Tofacitinib group. Following therapy, the VIDA score dropped to +3, +2, or +1.After the Baricitinib therapy, 13 (65.0%) of the 20 patients noted stopping disease progression, and after the Tofacitinib therapy, 11 (55.0%) of the 20 patients noted stopping disease progression. Repigmentation had appeared in 14 (70.0%) patient after Baricitinib therapy and 12 (60.0%) patients after Tofacitinib therapy. Response was excellent (≥75% repigmentation) in 6 patients, good (50%–75% repigmentation) in 4 patients, and moderate (25%–50% repigmentation) in 3 patients, while 1 patients showed a poor (<25% pigmentation) response in Baricitinib group and response was excellent (≥75% repigmentation) in 4 patients, good (50%–75% repigmentation) in 4 patients, and moderate (25%–50% repigmentation) in 2 patients, while 2 patients showed a poor (<25% pigmentation) response in Tofacitinib group.* ***Conclusion:*** *Although baricitinib and tofacitinib are both potential treatments for vitiligo, baricitinib may be a better choice, particularly when used with phototherapy.*

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INTRODUCTION

Vitiligo is an acquired, idiopathic autoimmune illness characterized by patchy depigmentation of the skin, hair, or both[1]. About 0.5–2% of adults and children worldwide suffer from the condition, which manifests as amelanotic, milky white, and clearly defined macules or patches encircled by healthy skin[2]. Vitiligo patients may experience stigmatization, which can have a detrimental effect on their mental health and quality of life, particularly for individuals with darker skin tones[3–5]. Phototherapy and systemic glucocorticoids are examples of conventional therapeutic approaches. A generally effective treatment for vitiligo is still elusive because to the lack of a comprehensive understanding of its pathophysiology, despite the testing of novel therapeutic approaches in clinical trials[1, 6–8].

Vitiligo is characterized by a gradual loss of melanocytes, which results in depigmentation[8]. Melanocyte-specific CD8+ T lymphocytes primarily infiltrate the dermal-epidermal interface next to melanocytes at the border of depigmented lesions and contribute to the removal and killing of melanocytes, according to compelling evidence from both in vivo and ex vivo studies[9–12]. The primary cytokine generated by CD8+ T cells, IFN-g, is essential to the pathophysiology of the illness[13]. The T cell chemokine receptor (CXCR3) and its many ligands, CXCL9, CXCL10, and CXCL11, are among the IFN-g-induced genes whose expression is elevated in depigmented skin lesions.

Other findings, such as the enriched infiltration of CXCR3+ CD8+ T cells, including melanocytespecific CD8+ T cells, in biopsies of vitiligo lesions and the elevated CXCR3 receptor expression on melanocyte-specific T cells in the blood and

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skin of vitiligo patients, are in line with the expression of genes induced by IFN-g[14–18].

The IFN-g-chemokine axis and its related positive feedback loop have been identified as a possible mechanism in the onset and progression of vitiligo based on numerous investigations carried out in animal vitiligo models. IFN-g, which is produced by autoreactive CD8+ T cells, encourages depigmentation. In order to attract more melanocyte-reactive T cells, IFN-g concurrently induces keratinocytes to produce CXCR3, which binds to CXCL9. Furthermore, through the CXCR3 receptor, CXCL10 attracts T cells to the skin, exacerbating and prolonging pre-existing vitiligo lesion[15, 19–21].

Neutralization of IFN-g antibodies stops CD8+ T cell accumulation and lesion depigmentation in mouse vitiligo models. It has been demonstrated that JAK inhibitors decrease IFN-g signaling, which helps vitiligo sufferers repigment. The three most widely known JAK inhibitors used to treat vitiligo are tofacitinib (Pfizer, New York, NY, USA), ruxolitinib (Celgene, Summit, NJ, USA), and baricitinib (Indianapolis, IN, USA).

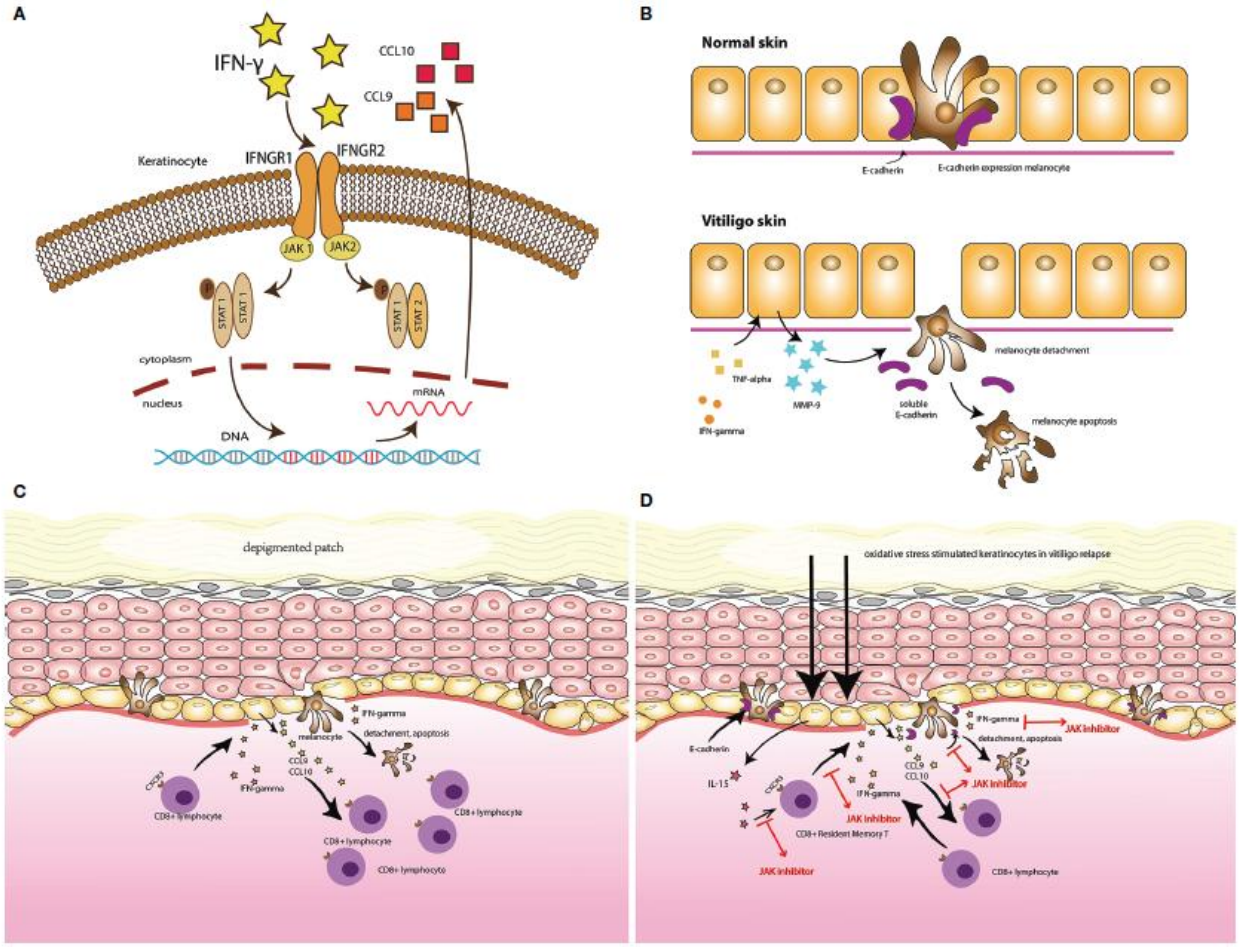

Figure I: (A) IFN- g signaling and the Janus kinase (JAK)/signal transducer and activator of transcription (STAT) pathway in vitiligo. (B) The secretion of MMP-9 by keratinocytes, in response to IFN-g and TNF-a, induced melanocytes detachment through E-cadherin disruption and released its soluble form, in the vitiligo skin compared with the normal skin. (C) Illustrates the vitiligo pathogenesis: the IFN-g-chemokine axis, with its associated positive-feedback loop: IFN-g, which is produced by autoreactive CD8+ T cells, encourages depigmentation. At the same time, it stimulates keratinocytes to express CXCR3, which binds to CXCL9 to attract more melanocyte-reactive T cells. Additionally, the CXCR3 receptor attracts T cells to the skin via CXCL10, worsening and prolonging the existing vitiligo lesions. (D) This highlights the potential use of JAK inhibitors for vitiligo treatment.

METHODS & MATERIALS

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

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window-based computer software program devised with Statistical Packages for Social Sciences (SPSS-24).

RESULT

Table – I: Distribution of the patients according to age (*n* = 40)

Table I shows that, Mean±SD of the patients age was 32.2 ±1.3 years and 38.6 ±2.3 years in Baricitinib and Tofacitinib group respectively

Age (years)	Baricitinib (n=20)	Tofacitinib (n=20)
21 – 25	3 (15.0)	2 (10.0)
26 – 30	4 (20.0)	4 (20.0)
31 - 35	7 (35.0)	3 (15.0)
36 - 40	4 (20.0)	8 (40.0)
41 - 45	2 (10.0)	3 (15.0)
Mean ± SD	32.2 ±1.3	38.6 ±2.3

Table – II: Distribution of the patients according to Sex (*n* = 40)

Table II shows that, most of the patients 11 (55.0%) were male in Baricitinib group and most of the patients 13 (65.0%) were female in Tofacitinib group

Sex	Baricitinib (n=20)	Tofacitinib (n=20)
Male	11 (55.0)	7 (35.0)
Female	9 (45.0)	13 (65.0)

Table – III: Distribution of the patients according to disease duration (*n*=40)

Table III Shows that, Mean±SD of disease duration was 6.2 ±1.3 years and 9.6±2.3 years in Baricitinib and Tofacitinib group respectively

Disease duration (years)	Baricitinib (n=20)	Tofacitinib (n=20)
1 – 3	3 (15.0)	2 (10.0)
4 – 6	4 (20.0)	4 (20.0)
7 - 9	7 (35.0)	3 (15.0)
10 - 12	4 (20.0)	8 (40.0)
Mean ± SD	6.2 ±1.3	9.6 ±2.3

Table – IV: Distribution of the patients according to treatment duration (*n* = 40)

Table IV shows that, Mean ± SD of treatment duration was 2.2 ±1.3 months and 3.6 ±2.1 months in Baricitinib and Tofacitinib group respectively

Treatment duration (month)	Baricitinib (n=20)	Tofacitinib (n=20)
2 – 3	7 (35.0)	8 (40.0)
4 – 5	4 (20.0)	4 (20.0)
6 - 7	3 (15.0)	3 (15.0)
8 - 9	4 (20.0)	2 (10.0)
Mean ± SD	2.2 ±1.3	3.6 ±2.1

Table – V: Distribution of the patients according to VIDA score (*n* = 40)

Table V shows that, VIDA score decreased to +3, +2, or +1 after therapy

Treatment duration (month)	Baricitinib (n=20)	Tofacitinib (n=20)
VIDA score at baseline	4	4
VIDA score After therapy	2	3

Table – VI: Distribution of the patients according to Arrest of progression (*n* = 40)

Table VI shows that, after the Baricitinib therapy, 13 (65.0%) of the 20 patients noted stopping disease progression and after the Tofacitinib therapy, 11 (55.0%) of the 20 patients noted stopping disease progression

Arrest of progression	Baricitinib(n=20)	Tofacitinib (n=20)
Yes	13 (65.0)	11 (55.0)
No	7 (35.0)	9 (45.0)

Table – VII: Distribution of the patients according to Repigmentation (*n* = 40)

Table VI Ishows that, repigmentation had appeared in 14 (70.0%) patient after Baricitinib therapy and 12 (60.0%) patients after Tofacitinib therapy

Repigmentation	Baricitinib (n=20)	Tofacitinib (n=20)
Yes	14 (70.0)	12 (60.0)
No	6 (30.0)	8 (40.0)

Table VIII shows that, Six patients had excellent (≥75%) repigmentation, four had good (50–75%) repigmentation, and three had moderate (25%–50%) repigmentation; one patient had a poor response. (<25% pigmentation) response in Baricitinib group and response was excellent (≥75% repigmentation) in 4 patients, good (50%–75% repigmentation) in 4 patients, and moderate (25%–50% repigmentation) in 2 patients, while 2 patients showed a poor (<25% pigmentation) response in Tofacitinib group.

Table – VIII: Distribution of the patients according to outcome (*n* = 40)

Outcome	Baricitinib (n=14)	Tofacitinib (n=12)
Excellent	6 (30.0)	4 (20.0)
Good	4 (20.0)	4 (20.0)
Moderate	3 (15.0)	2 (10.0)
Poor	1 (5.0)	2 (10.0)

DISCUSSION

A characteristic of vitiligo, a depigmenting skin disorder that results in pigment dilution in the affected skin areas, is

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selective melanocyte loss. A chalky-white, nonscaly, amelanotic macule with prominent edges is the hallmark lesion. The pathophysiology of vitiligo has been better understood in recent years, and it is now firmly established that it is an autoimmune disease with metabolic, environmental, and genetic causes, as well as oxidative stress and cell detachment. Vitiligo should not be dismissed as a minor or cosmetic condition because it can have profoundly disruptive effects on everyday life and have mentally devastating consequences.

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, Mean± SD of the patients age was 32.2±1.3 years and 38.6 ±2.3 years in the Baricitinib and Tofacitinib group respectively. Most of the patients 11 (55.0%) were male in Baricitinib group and most of the patients 13 (65.0%) were female in Tofacitinib group. Mean ± SD of disease duration was 6.2±1.3 years and 9.6±2.3 years in Baricitinib and Tofacitinib group respectively. Mean ± SD of treatment duration was 2.2±1.3 months and 3.6±2.1 months in Baricitinib and Tofacitinib group respectively. VIDA score decreased to +3, +2, or +1 after therapy. After the Baricitinib therapy, 13 (65.0%) of the 20 patients noted stopping disease progression and after the Tofacitinib therapy, 11 (55.0%) of the 20 patients noted stopping disease progression. Repigmentation had appeared in 14 (70.0%) patient after Baricitinib therapy and 12 (60.0%) patients after Tofacitinib therapy. Six patients had excellent (≥75%) repigmentation, four had good (50–75%) repigmentation, and three had moderate (25%–50%) repigmentation; one patient had a poor response (<25% pigmentation) response in Baricitinib group and response was excellent (≥75% repigmentation) in 4 patients, good (50%–75% repigmentation) in 4 patients, and moderate (25%–50% repigmentation) in 2 patients, while 2 patients showed a poor (<25% pigmentation) response in Tofacitinib group.

An approved treatment for moderate to severe rheumatoid arthritis is tofacitinib, a selective JAK1 and JAK3 inhibitor[22]. Tofacitinib, both topical and oral, has demonstrated effectiveness in treating alopecia areata, atopic dermatitis, and plaque psoriasis, among other immune-mediated skin conditions[23–25]. Tofacitinib was initially administered orally to a female patient with vitiligo who had about 10% depigmentation in her entire body surface area and who did not respond to topical corticosteroid and tacrolimus ointments. The patient was provided 5 mg of oral tofacitinib citrate on alternate days, which was raised to 5 mg daily starting in week four due to the suspected shared pathophysiology of vitiligo and alopecia areata.

After five months of treatment, only 5% of the patient's total body surface area remained depigmented. Throughout the course of treatment, no adverse effects were noted[26].

In a retrospective study, changes in the autoimmune responses of ten vitiligo patients on tofacitinib were evaluated using suction blister sampling. Ten patients received tofacitinib therapy at a dose of 5–10 mg daily or twice daily for an average of 9.9 months; only half of the patients had re-pigmentation in sun-exposed or phototherapy-only areas. Following tofacitinib treatment, flow cytometry showed a decrease in CD8+T cells but no change in the proportion of T cells specific to melanocytes. Furthermore, following tofacitinib treatment, chemokines such CXCL9 and CXCL10 decreased and were no longer detectable[27].

In line with the discovery that sun-exposed regions, like the hands and face, respond better to topical ruxolitinib treatment[28], these results suggest that re-pigmentation of vitiligo lesions may require both JAK inhibitors (to inhibit local inflammation) and light exposure (to stimulate melanocytes)[28]. Additionally, 16 individuals with vitiligo, including 11 with widespread vitiligo, received topical 2% tofacitinib cream.

In line with earlier research, individuals with darker skin tones and face lesions showed more pronounced responses, but concurrent phototherapy did not produce any better outcomes, which runs counter to earlier findings[29]. Tofacitinib's use in treating vitiligo is not yet being studied in any registered clinical studies; more study is required to ascertain its safety and effectiveness as well as the function of phototherapy when combined with tofacitinib.

Only one case report has documented vitiligo lesions treated with baricitinib, a particular inhibitor of JAK1 and JAK2[22]. A 67-year-old man with vitiligo on his hands and forearms experienced complete repigmentation after switching from tofacitinib 5 mg twice daily to baricitinib 4 mg daily for the treatment of rheumatoid arthritis[30]. To assess the safety and effectiveness of baricitinib and phototherapy together in the treatment of vitiligo, new phase 2 research (NCT04822584) is in underway[31]. Baricitinib is a selective JAK1/2 inhibitor that is commonly used to treat vitiligo, alopecia areata, and atopic dermatitis in addition to rheumatoid arthritis. Since a few case studies have shown that baricitinib and phototherapy work well together to treat vitiligo, a new phase 2 trial (NCT04822584) is currently underway to evaluate the safety and efficacy of this combination[15, 30]. In this trial, we used oral baricitinib monotherapy to treat four young patients with vitiligo that was getting worse. One of them had vitiligous lesions on the trunk, while three others had vitiligous lesions on the face and neck. By the conclusion of week[12], all patients had improved clinically, with re-pigmentation rates of 59.26-74.17% and significantly lower VASI levels. Nevertheless, baricitinib showed a significant re-pigmentation potential, particularly in patients with vitiligous lesions on sun-exposure areas, which was in line with the findings of published literature, even though statistical analysis was not carried out in this patient group due to the small sample size[15, 31]. Every patient responded well to the treatment and experienced no serious adverse effects. Due to the fact that baricitinib was being used off-label, the therapy ceased after 12 weeks.

CONCLUSION

Although baricitinib and tofacitinib are both potential treatments for vitiligo, baricitinib may be a better choice, particularly when used with phototherapy. However, there

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are possible hazards associated with both medications, so choosing one should be discussed with a healthcare provider.

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