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(REVIEW ARTICLE)

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Pharmacotherapy of atopic dermatitis: a comparison of clinical response with Lebrikizumab and Tezepelumab

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Abstract

Atopic dermatitis is a disease that affects millions worldwide. Research indicates that people suffering from atopic dermatitis respond well to monoclonal antibody treatment aimed at characteristic immune cell markers. By targeting immune cell markers, the immune system is subsequently suppressed in a way such that disease activity is decreased. Monoclonal antibody treatments targeted towards a variety of immune cell markers have historically been shown to result in decreases in atopic dermatitis disease activity. Many studies have assessed disease activity with monoclonal antibody treatments, but few have compared different treatments.

A systematic review of a study that assessed the disease activity for those taking tezepelumab was then compared to the findings of another study which assessed the disease activity with those taking lebrikizumab.

The patients treated with tezepelumab had significantly decreased atopic dermatitis activity when compared with other matched controls. Those who were treated with lebrikizumab had significantly decreased disease activity when compared with other matched controls. However, those who were treated with lebrikizumab had a greater reduction of disease activity as compared to control groups than did those who were treated with lebrikizumab.

Monoclonal antibody treatments for atopic dermatitis are widespread. It is critical that these treatments are compared in order to find those with the greatest efficacy. For this review, lebrikizumab seemed to have a greater reduction of disease activity than did tezepelumab. Further studies with greater sample sizes are needed to conclude which has greater efficacy

Keywords: Atopic dermatitis; Tezepelumab; lebrikizumab; Autoimmune Disease; Monoclonal Antibody Therapy; Inflammatory Skin Disease

1. Introduction

There is sufficient information to suggest that persons suffering from atopic dermatitis respond well to monoclonal antibody therapy (1). Two prominent monoclonal antibody treatments for the treatment of atopic dermatitis are the anti–thymic stromal lymphopoietin agents such as tezepelumab and the anti-interleukin-13 (anti-IL-13) agents such as lebrikizumab (1,2).

Anti–thymic stromal lymphopoietin agents such as tezepelumab are directed towards anti–thymic stromal lymphopoietin, which is a cytokine that is implicated in the pathogenesis of atopic dermatitis (1). IL-13 inhibitors such as lebrikizumab inhibit the IL-13-mediated cascade which is believed to play a key role in type 2 inflammation and is an emerging pathogenic mediator in atopic dermatitis (2).

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It is well known that both of these monoclonal antibody therapies have significant effects in increasing the amount of participants that reached a ≥50% reduction in the Eczema Area and Severity Index (EASI50) after 12 weeks, but little has been done to compare their efficacies (1,2). This is especially important to know such that a physician can prescribe the best pharmacological intervention to treat a patient with atopic dermatitis.

2. Methods

For the study conducted using tezepelumab (1):

In this phase 2a study, 113 patients were randomized 1:1 to subcutaneous tezepelumab 280 mg or placebo every 2 weeks, plus class 3 topical corticosteroids (TCS). The primary endpoint was the week 12 response rate for a ≥50% reduction in the Eczema Area and Severity Index (EASI50). Secondary endpoints including EASI75, Investigator's Global Assessment, SCORAD 50, SCORAD 75, pruritus numeric rating and 5-D itch scales, and exploratory endpoints (including EASI90) were assessed at weeks 12, and 16 (post hoc).

For the study conducted using lebrikizumab (2):

This was a randomized, placebo-controlled, double-blind, phase 2 study, a total of 209 patients received the study drug. Adults with moderate-to-severe AD were required to use TCS twice daily and then randomized (1:1:1:1) to lebrikizumab 125 mg single dose, lebrikizumab 250 mg single dose, lebrikizumab 125 mg every 4 weeks for 12 weeks, or placebo every 4 weeks for 12 weeks, after a 2-week TCS run-in. The primary endpoint was percentage of patients achieving Eczema Area and Severity Index (EASI)-50 at week 12.

3. Results

For the study conducted using tezepelumab (1):

A numerically greater percentage of tezepelumab plus TCS-treated patients achieved EASI50 (64.7%) versus placebo plus TCS (48.2%; p = .091). Numerical improvements over placebo were demonstrated for week 12 secondary and exploratory endpoints, with further improvements at week 16. Treatment-emergent adverse events were similar between treatment groups.

For the study conducted using lebrikizumab (2):

At week 12, significantly more patients achieved EASI-50 with lebrikizumab 125 mg every 4 weeks (82.4%; p = .026) than placebo every 4 weeks (62.3%); patients receiving a single dose of lebrikizumab showed no statistically significant improvements in EASI-50 compared with placebo. Adverse events were similar between groups (66.7% all lebrikizumab vs 66.0% placebo) and mostly mild or moderate.

Table 1 EASI-50 Results for Both Treatments

Treatment	Percent Achieving EASI-50 (%)	p-value
lebrikizumab 125mg	82.5%	0.026
tezepelumab 280mg	48.2%	0.091

The above table (**Table 1**) depicts the percentage achieving EASI-50 and p-values for each of the treatment groups in each study. Groups treated with lebrikizumab 125mg achieved statistically significant increases in EASI-50 whereas those treated with tezepelumab 280mg did not

4. Conclusion

Our analysis suggests that while both tezepelumab and lebrikizumab might be effective in reducing atopic dermatitis disease activity, tezepelumab increased the EASI-50 by more than 30% that of the tezepelumab-treated group. This also indicates that interleukin-13 is likely more implicated in atopic dermatitis than the anti–thymic stromal lymphopoietin. The analyses are limited in the sense that they both couldn't accurately control for the severity of atopic dermatitis and

comorbidities. The dosage between each study could not be controlled, nor could the potency be quantified. There is also the likelihood that there is another immune marker that may be even more implicated in the pathogenesis of atopic dermatitis than anti–thymic stromal lymphopoietin and interleukin-13. Also, further studies must be conducted with larger sample sizes controlling for age and other demographic factors. Since both agents were in phase II trials, the therapeutic index of either has likely not been established. While lebrikizumab shows promising results, there is plenty more investigation that must be conducted to find the most effective monoclonal antibody treatment for atopic dermatitis

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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