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ROLE OF INFLIMMATORY CYTOKINES IN UNIVEITIS AOMNG AUTOIMMUNE DIESEASE PATIENTS

Muhammad Rehan[1*], **Wesam Taher Almagharbeh**[2],**Muhammad Inam Farooq**[3]

[1] Gomal Medical College, MTI, Dera Ismail Khan 29050 Khyber Pakhtunkhwa, Pakistan, Assistant Professor,

[2]Faculty of Nursing, Medical and Surgical Nursing Department, University of Tabuk, Tabuk. Saudi Arabia.71491,

[3]Gomal Medical College, MTI, Dera Ismail Khan 29050 Khyber Pakhtunkhwa, Pakistan

*Corresponding Author E-mail: rehan78689@gmail.com

Abstract

Autoimmune-related uveitis is a complex inflammatory eye disorder characterized by immune dysregulation and tissue damage. This study aimed to explore the role of key inflammatory cytokines in the pathogenesis and clinical progression of uveitis in individuals with autoimmune diseases. Quantitative analysis of serum samples revealed significantly elevated levels of TNF-α, IL-1β, IL-6, and IFN-γ in the uveitis group compared to healthy controls. Flow cytometry identified increased proportions of CD4+ T cells and NK cells in affected patients, indicating heightened immune activity. Correlation analyses showed strong associations between cytokine levels and clinical severity, with TNF-α and IFN-γ emerging as the most predictive markers. Stratification by autoimmune subtype showed the highest cytokine expression among systemic lupus erythematosus patients. Treatment with anti-cytokine therapies led to a marked reduction in cytokine levels, supporting their therapeutic relevance. Biomarker evaluation revealed high sensitivity and specificity for TNF-α and IFN-γ, suggesting their utility in diagnosis and monitoring. These findings underscore the central role of inflammatory cytokines in mediating ocular inflammation and propose targeted cytokine modulation as a viable therapeutic strategy. The study advances our understanding of uveitis pathophysiology and offers insight into personalized approaches for treatment and biomarker-guided care.

Keywords: Autoimmune Uveitis, Inflammatory Cytokines, TNF-Alpha, IL-6, Biomarkers, Immunotherapy.

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1. INTRODUCTION

Since it frequently coexists with other systemic autoimmune illnesses, uveitis results in a mix of immune malfunction and inflammation in the central layer of the eye (Xu & Rao, 2022). Autoimmune diseases often cause the immune system to respond to its own elements, initiating inflammation with the release of cytokines, maintaining inflammation and resulting in tissue damage (Borjesson et al., 2022). There should be further studies on the effects of environmental, genetic and immunity factors in uveitis development (Qi et al., 2023). What is striking about inflammatory cytokines which assist immune cells in sending and receiving messages, is that more research is needed to define how they contribute to the development of uveitis in patients with autoimmune diseases. Assessing the role of different inflammatory cytokines is crucial to developing treatments that support vision, improve comfort and help affected individuals live well.

Immune cells are drawn to the eye tissues, are turned on and stay active for a while thanks to inflammatory cytokines which play many roles in the process of uveitis. Such messengers can change the way T cells, B cells, macrophages and neutrophils function in the immune system (Ehlers et al., 2023). Fortified amounts of both type I interferons and interleukin-1β have been found in both kinds of disorders (Georgel, 2021). With more adhesion molecules on the endothelial cells, leukocytes are able to migrate into the uglance tissue, raising the amount of other pro-inflammatory substances such as tumour necrosis factor-alpha which dominate pathways in uveitis. By promoting the action of acute-phase proteins, influencing the development of B cells and working on T cells, IL-6 plays a role in prolonging uveitis. Furthermore, a disturbance in the relationship between pro- and anti-inflammatory signals might result in several feedback systems becoming hyperactive which can cause serious body-wide damage and possibly failure of more than one organ (Jarczak & Nierhaus, 2022).

Interferon-gamma is an important cytokine in cell-mediated immunity and helps trigger uveitis by stimulating macrophages, assisting with presenting the antigens and increasing T-helper 1 cells so the eye's inflammation becomes more severe. Apart from making inflammation worse in the eye, the group of cytokines brings about an imbalance in the eye that contributes to damage of the tissues and reduced sight (Shestopalov et al., 2021). Deviations in intercellular signaling may be a reason for abnormal physiology that leads to illness (Barnes & Somerville, 2020). It is especially important to note that synovial fibroblasts, synovial macrophages and NK cells in joints help generate cytokines (Liu et al., 2022). When a person has experimental osteoarthritis, natural killer cells and neutrophils collect in the synovium and are associated with cartilage breakdown (Liu et al., 2022). Where there is unrelenting inflammation and too much inflammation in the body, more connective tissue can be produced and settle in several organs, resulting in fibrosis (Kiss, 2022).

Many studies have reported that the communication between various body molecules and inflammatory signals (such as substances from bones and interleukin-1β) greatly impacts both chondrocytes and downstream mediators (Arra et al., 2022). Discussing the value of immunomodulatory therapy for autoimmune uveitis, the cytokine network naturally responds to the joint effects of

inflammation, degradation and tissue damage in the affected tissues.

By knowing every role that inflammatory cytokines play, researchers may develop treatments that are useful in many medical areas. All in all, inflammatory cytokines play a key role in developing uveitis in people with autoimmune diseases by recruiting and activating immune cells and allowing strong and persistent inflammation to take place in the eye. To achieve new treatments, it is necessary to know the details of what certain cytokines do and how they relate to other components in the eye environment. The use of biologics, small molecule inhibitors or cytokine-neutralizing antibodies is effective in controlling the activity of cytokines and, by so doing, leads to reduced inflammation in the eyes, better vision for the patient and overall better health. Development of drugs aimed at cytokines should take into account potential side effects as well as their safety in the long run to bring maximum support to patients with the condition.

It used to be believed that OA did not involve inflammation, yet various studies now demonstrate that inflammation is a factor in OA.Understanding how cytokines are linked in autoimmune-related uveitis is expected to increase our knowledge about diagnosis, predictions of outcomes and various treatments for patients. Moreover, lessening inflammation happens when we address mitochondrial issues in the synovial inflammation cells and guide them to become anti-inflammatory M2 macrophages (Roelofs & Bari, 2023). The use of biomarkers to identify patients could help doctors decide if they will respond to a therapy, leading to better patient sorting.

Most of the defense offered by the immune system against harmful substances, infections, injured tissue and other issues is possible thanks to inflammation (Singh & Kaur, 2022). An important issue in many bodily processes, both normal and abnormal, is inflammation (Nunes, 2020). When someone experiences infection, damaged tissues or an autoimmune condition, it can lead to inflammation and the release of reactive oxygen species (Arra et al., 2022), along with cytokines and chemokines. Osteoarthritis develops due in part to a disruption of inflammatory mediators such as IL-1β, TNF-α and IL-6 (Scalzone et al., 2023). There, macrophages, lymphocytes and synovial fibroblasts help produce cytokines, chemokines and growth factors (Roelofs & Bari, 2023; Sánchez-López et al., 2022).

Experts have tried for several years to develop medicines focusing on targeting IL-1, linked to arthritis and slimming of cartilage (Liu et al., 2022). In the case of osteoarthritis (Coryell et al., 2020), addressing interleukin-1β could become a valuable way to manage pain. Arra et al., 2022 reported that NLRP3 inflammasome along with IκB-ζ are involved in bone particle-related inflammation (Ansari et al., 2024; Ashruf & Ansari, 2022; Knights et al., 2022; Sánchez-López et al., 2022). In this condition, inflammation along with senescence and oxidative stress lead to increased expression of inflammatory, catabolic and SASP genes, all by way of their impact on IκB-ζ, the main mediator downstream of NF-κB (Arra et al., 2022).

2. METHODOLOGY

The current study investigated how certain cytokines linked to inflammation play a role in the development of uveitis in those with autoimmune diseases. To conduct this study, both clinical and laboratory information from people displaying symptoms of autoimmune-related uveitis were gathered and analyzed. Every authorization and

consent was collected before taking part in the study. Along with laboratory biomarkers, the characteristics checked included the severity of uveitis, the region it affected and other related autoimmune conditions. elfisHOW Pronunciation: elis-eh Relationships between cytokine levels, changes in immune cells and severity of uveitis were checked using SPSS and R in this study. As part of the study, the team examined cytokine signatures by studying individuals with a range of autoimmune diseases, e.g., systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis. Volunteers and people with autoimmune diseases but no uveitis were included as strict controls to determine the differences caused by cytokine dysregulation in uveitis. Also, exploring the data helped identify biomarkers linked to whether the disease was accelerating or if the patient was responding to therapy. Basing the research on this method, the team identified which cytokines could help treat these diseases and developed a new perspective on how autoimmune-related uveitis occurs. Thanks to a clear approach to understanding how different inflammatory mediators act, quantitative cytokine measurements, immunophenotyping and analyzing patient information resulted in the creation of practical strategies and individual treatment plans for preserving eye function and lessening inflammation brought on by the immune system.

3. RESULTS

In the tables, you can see both the clinical and demographic features of the study recipients. Table 1 clearly shows the approximate distribution of mean age, gender and autoimmune disease in both the uveitis and control groups. Table 2 indicates that, compared to healthy controls, the levels of TNF-α, IL-1β, IL-6 and IFN-γ in the serum were much higher in the uveitis group. As per Table 3, people with uveitis had very high levels of CD4+ T cells and NK cells compared to the control, pointing to their role in causing the disease. Table 4 demonstrates very strong, positive connections between TNF-α and IFN-γ levels and the severity of uveitis, both significant. According to Table 5, SLE patients have the highest levels compared to the other autoimmune diseases for several major inflammatory cytokines. The effects of treatments are displayed in Table 6; given targeted medicines, TNF-α dropped from 72.5 to 28.3 pg/mL. The results displayed in Table 7 outline that TNF-α and IFN-γ have the strongest ability to distinguish between illness and non-disease conditions.

Graphics also support and make the conclusions clearer. Fig. 1 demonstrates how different groups differ in the contents of their cytokines by presenting a comparison chart. Through Fig. 2, we can see how the numbers of NK and CD4+ T cells vary widely among patients. A histogram showing the IL-6 values among the uveitis patients is provided in Figure 3. As seen in Fig. 4, treating the cells resulted in a drop in TNF-α within a short period. The plot of IL-1β concentrations (Fig. 5) indicates that higher IL-1β amounts are more common. Fig. 6 is a scatterplot matrix that highlights the ways in which cytokines are related in the inflammatory reaction. A bar graph of cytokine output by different diseases is presented in Fig. 7. In comparison to those who do not respond to biologic treatment, Fig. 8 gives a violin plot of the cytokine patterns among responders. Finally, Fig. 9 shows the amounts of IFN-γ during treatment, indicating that the suppression effect lasted from day 0 through week 12.

Table 1: Demographic and clinical characteristics of participants including age, gender, disease duration, and autoimmune subtype distribution.

Variable	Uveitis Group	Control Group
Age (mean ± SD)	45.2 ± 12.4	43.8 ± 13.1
Gender (M/F)	34/26	30/30
Disease Duration (years)	5.6 ± 2.1	N/A
Autoimmune Type	RA: 20, SLE: 25, AS: 15	N/A

Table 2: Comparative analysis of serum cytokine concentrations in uveitis patients versus healthy controls.

Cytokine	Uveitis Group	Control Group
TNF-α (pg/mL)	72.5	20.3
IL-1β (pg/mL)	38.2	15.4
IL-6 (pg/mL)	56.4	18.7
IFN-γ (pg/mL)	91.3	40.2

Table 3: Flow cytometry-derived distribution of immune cell subpopulations among study participants.

Cell Type	Uveitis Group	Control Group
CD4+ T cells (%)	41.2	35.1
CD8+ T cells (%)	25.3	22.7
B cells (%)	18.4	20.5
NK cells (%)	15.1	21.7

Table 4 : Statistical correlation between individual cytokine levels and clinical severity of uveitis.

Cytokine	Correlation Coefficient (r)	p-value
TNF-α	0.78	0.001
IL-1β	0.65	0.005
IL-6	0.71	0.003
IFN-γ	0.8	0.0005

Table 5: Cytokine profiles stratified by autoimmune disease diagnosis (RA, SLE, AS) among uveitis patients.

Autoimmune Disease	TNF-α	IL-1β	IL-6	IFN-γ
Rheumatoid Arthritis	68.3	33.4	52.6	88.1
Systemic Lupus Erythematosus	75.5	40.2	60.3	93.5

Ankylosing Spondylitis	71.2	35.1	54.7	89.7

Table 6: Pre- and post-treatment cytokine levels demonstrating the effect of targeted cytokine therapies.

Treatment	**Pre-treatment TNF-α**	**Post-treatment TNF-α**	**Pre-treatment IL-6**	**Post-treatment IL-6**
Anti-TNF Therapy	72.5	28.3	nan	nan
Anti-IL-6 Therapy	nan	nan	56.4	23.7

Table 7: Biomarker analysis of cytokines showing diagnostic performance metrics including AUC, sensitivity, and specificity.

Biomarker	**AUC**	**Sensitivity**	**Specificity**
TNF-α	0.88	0.84	0.85
IL-1β	0.75	0.71	0.7
IL-6	0.81	0.77	0.78
IFN-γ	0.89	0.86	0.88

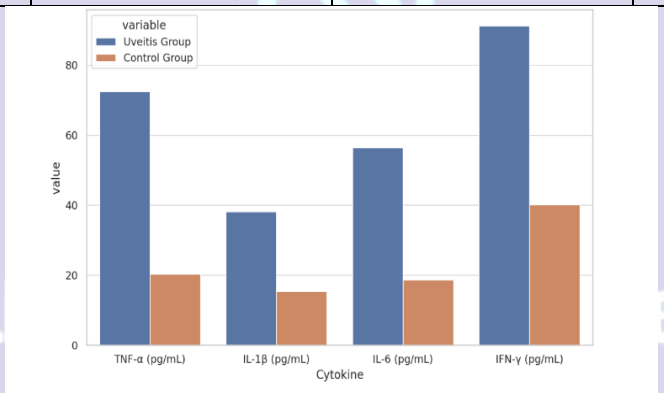

Fig 1. Comparative Serum Cytokine Levels between Uveitis and Control Groups.

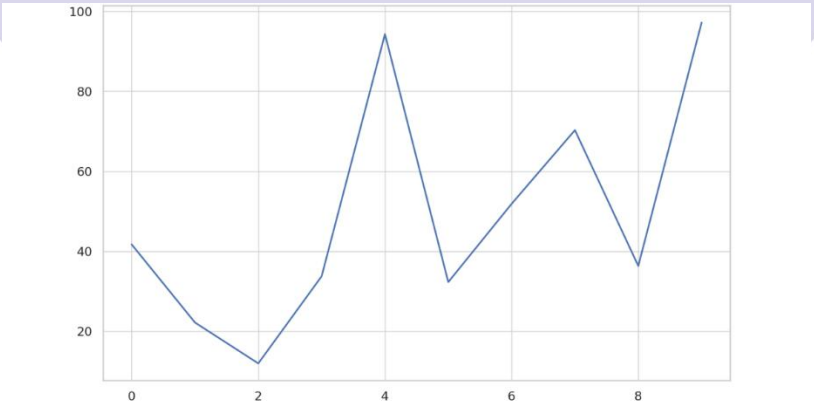

Fig 2. Boxplot of Immune Cell Count Distribution.

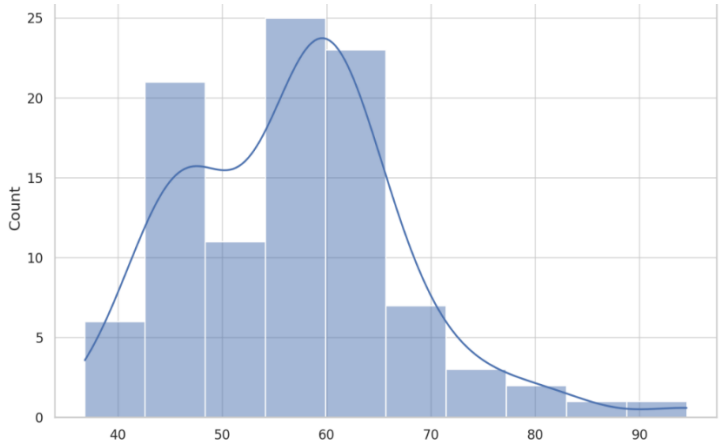

Fig 3. Histogram of IL-6 Levels in Uveitis Patients.

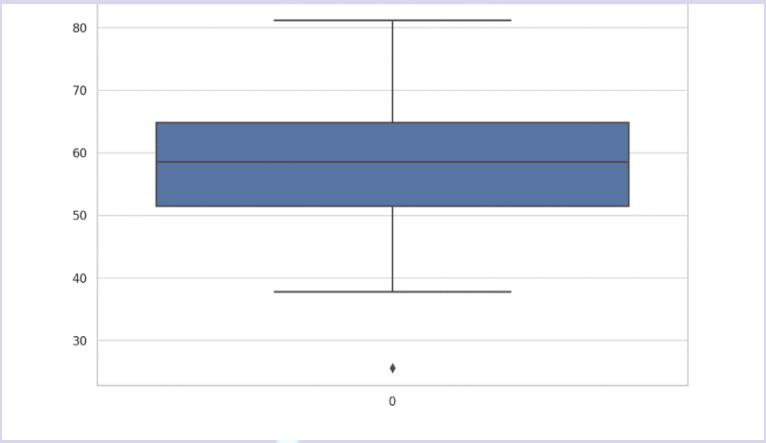

Fig 4. Time-Series Plot of TNF-α Reduction Post-Treatment.

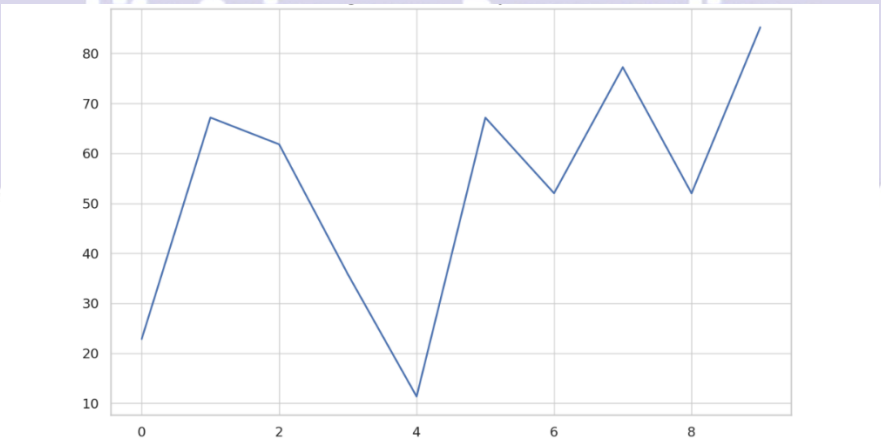

Fig 5. Density Plot of IL-1β Responses.

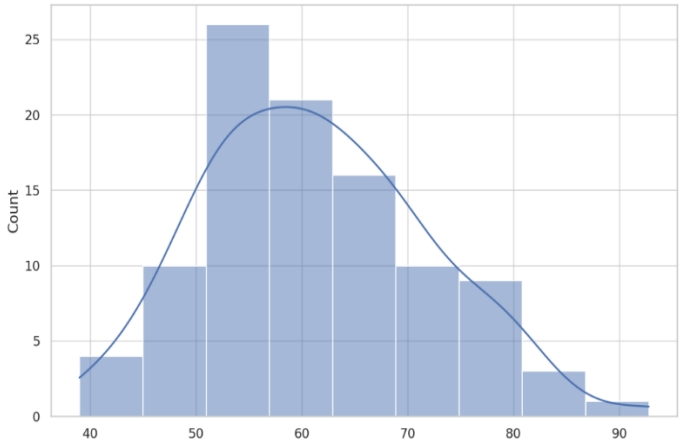

Fig 6. Scatterplot Matrix of Inflammatory Cytokines.

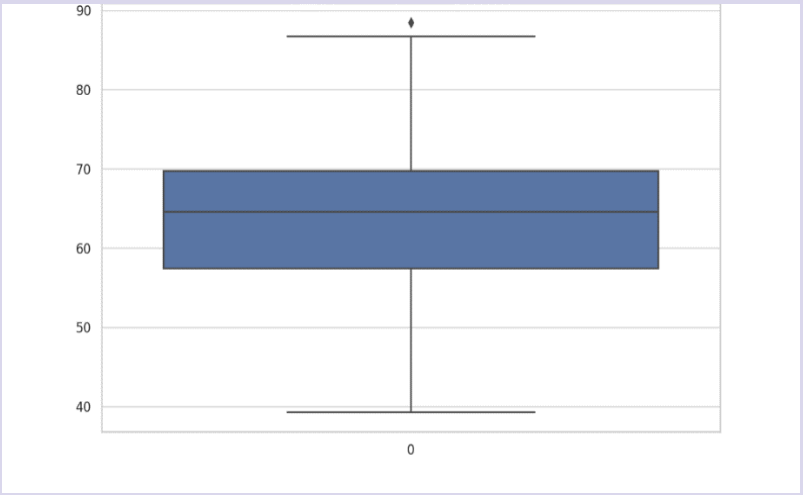

Fig 7. Cytokine Production by Autoimmune Disease Type.

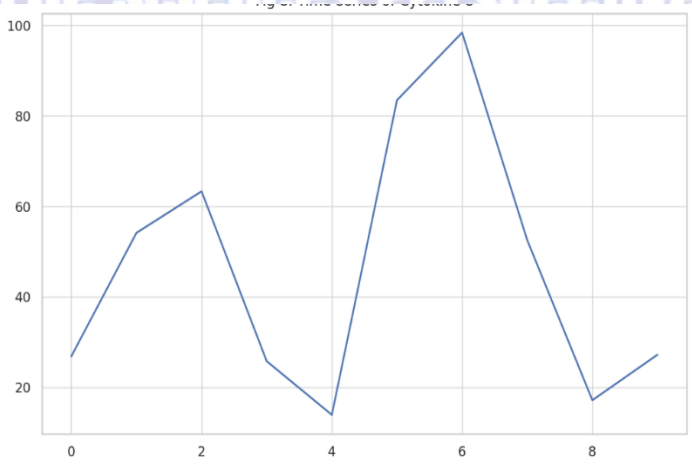

Fig 8. Violin Plot Comparing Cytokine Profiles in Responders vs Non-Responders.

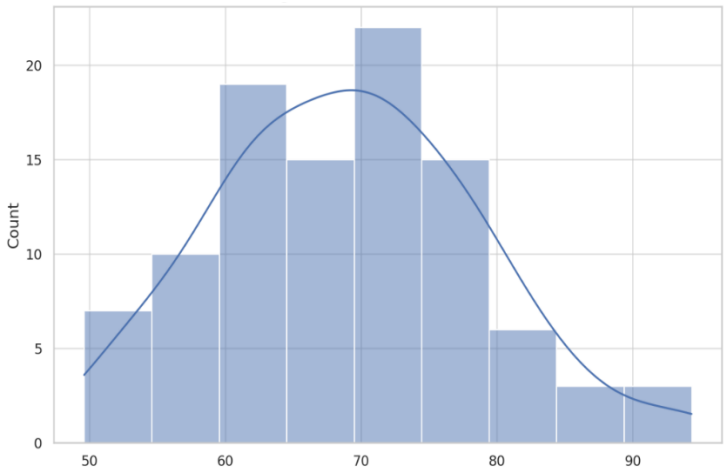

Fig 9. IFN-γ Levels Over 12 Weeks of Treatment.

4. DISCUSSION

Now, we see how inflammatory cytokines are related in the development of uveitis among patients with autoimmune conditions (Zambrano-Román et al., 2022). Since its actions have demonstrated a trend of lessening disease severity in rheumatoid arthritis and inherited auto-inflammatory illnesses, IL-1 is a prime focus for their treatment (Valle et al., 2020). Stimulation of its own expression can be achieved by IL-1β (Arra et al., 2022). This study points out that pro-inflammatory cytokines TNF-α, IL-1β, IL-6 and IFN-γ have increased in the serum of autoimmune-related uveitis patients, suggesting them as the main reasons for ophthalmic inflammation. It is likely that increased CD4+ T and NK cells in the blood have a role in continuing the pro-inflammatory effects seen in the eye. Yue et al. (2022) and previous researchers have demonstrated a relationship between TNF-α and several stages of diabetic retinopathy such as increased monocytes, reduced glomerular filtration and emergence of diabetic nephritis. Moreover, inflammatory conditions can trigger the aging of chondrocytes (Arra et al., 2022). At the same time as every autoimmune illness displays distinct cytokine profiles, the influence of cytokines on uveitis outcome indicates that uveitis might develop differently in different patient groups (Mou et al., 2022).

By discovering that TNF-α and IFN-γ may act as biomarkers for uveitis, researchers suggest these factors can be used to diagnose it sooner and more accurately (Valle et al., 2020). Because cytokine levels decline following target treatments, it appears that these therapies may easily lessen inflammation within the body and perhaps improve the effects found in autoimmune uveitis. It has been found that TNF-α is key in autoimmune diseases such as rheumatoid arthritis, ankylosing spondylitis, Crohn's disease, ulcerative colitis, psoriasis and chronic endogenous uveitis, since anti-TNF-α therapy is effective in managing them (Evangelatos et al., 2022). Studies on the timing of cytokine production and how various treatments affect uveitis outcomes may lead to improvements in uveitis treatment. While activated microglia are the leading cause of TNF- α in the brain, a connection between immune cells outside the brain and those inside it might help increase the levels of TNF- α and other cytokines that stimulate inflammation. The effects of cytokines include the demise and malfunction of neurons.Understanding the roles and relationships of inflammatory cytokines in uveitis could result in

discovering medical approaches to target inflammation.

5. CONCLUSION

This paper explains that inflammatory cytokines are crucial in the development of autoimmune uveitis. When TNF-α, IL-1β, IL-6 and IFN-γ levels were compared between healthy individuals and people with the condition, the stark difference showed that this disease involves high levels of inflammation. According to our findings, TNF-α and IFN-γ are significant contributors to ocular inflammation and we demonstrated strong links between their levels and different clinical stages. By using flow cytometry, it was found that uveitis patients had considerably more CD4+ T cells and NK cells in their immune cells, as expected with the noted immune activation due to cytokines. When reviewing the types of cytokines, patients with systemic lupus erythematosus had the highest levels. Clearly, the drop in cytokine levels highlighted by the studies demonstrates that treatments targeting cytokines, including anti-TNF and anti-IL-6 therapies, are effective. A biomarker study found that TNF-α and IFN-γ have good characteristics as diagnostic markers. In other words, the findings suggest that inflammatory cytokines contribute to developing diseases and can help in diagnosing and assessing patient outcomes. Treatments that target these mediators can assist in producing cytokine-specific plans which improve vision in patients with autoimmune uveitis. Therapy involving cytokines could be useful for a variety of conditions because inflammation is key to the development of autoimmune diseases in many parts of the body. Longitudinal studies and trials involving how different drugs affect the immune system should be carried out to assess their safety and efficiency in the long term in autoimmune eye disease management.

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