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INFLAMMATORY PATHWAYS IN FIBROMYALGIA: A CYTOKINE PROFILING STUDY

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Abstract

Fibromyalgia is a multifactorial chronic pain disorder characterized by widespread musculoskeletal pain, fatigue, cognitive dysfunction, and mood disturbances. While central sensitization is widely accepted as a core mechanism, emerging evidence suggests that systemic inflammation may also play a crucial role in the pathophysiology of fibromyalgia. This study aimed to investigate the cytokine profile in fibromyalgia patients and its correlation with clinical symptom severityIncluded in the study were 20 healthy women referred to as controls and 40 patients with a fibromyalgia diagnosis. Using multiplex immunoassay, it was possible to measure the serum amounts of cytokines such as IL-1β, IL-6, IL-8, TNF-α, IFN-γ, IL-10 and TGF-β1. Included in the clinical assessments were the Visual Analogue Scale for pain, the Hospital Anxiety and Depression Scale and the Fibromyalgia Impact Questionnaire-Revised. The study investigated the relationships between levels of cytokines and the various symptoms. Levels of cytokines IL-6, IL-1β, IL-8, TNF-α and IFN-γ as well as TGF-β1 were much higher in patients with fibromyalgia than in controls. The patients with fibromyalgia were found to have lower anti-inflammatory IL-10 levels. The results showed reduced functioning (FIQR = 59.5), increased psychological stress (HADS-Anxiety = 11.5 and HADS-Depression = 10.1) and reported severe pain (VAS = 7.8). On the other hand, although IL-6 was closely linked to higher VAS and FIQR scores, IL-10 was negatively linked to a person's feelings of melancholy and anxiety.

Keywords: Fibromyalgia, Cytokines, Inflammation, IL-6, TNF-Alpha, Chronic Pain.

INTRODUCTION

Nowadays, fibromyalgia is no longer considered a mere debated disease, but a genuine neuropathic pain disorder brought to light through tiny fibre changes seen with ocular confocal and skin biopsies (Martínez-Lavín, 2021). However, since its exact causes cannot always be shown and existing neuropathic pain drugs do not relieve symptoms, fibromyalgia has not yet been defined strictly as a neuropathic condition (Evans et al., 2021). Consequently, it is important to research more thoroughly about possible alternative processes influenced by fibromyalgia, for instance systemic inflammation (Megha et al., 2021).

Analyzing the inflammatory factors involved with fibromyalgia might help reveal its many causes. Studies suggest that this condition may be linked to inflammation, caused by depressed quantities of certain cytokines. Having inflammation in the central nervous system is controlled by cytokines and can both serve as a means of protection and be involved in causing sickness (Mallick et al., 2025). In patients with fibromyalgia, since central sensitisation is an issue, long-term inflammation can cause nerves in the body to fire more often and make pain worse.

Since inflammation and nerve activity are closely related, substances released during inflammation might influence how a person with fibromyalgia perceives and processes pain. It is clear that studying inflammatory chemicals in this way affects the expression and role of several proteins such as those involved in chemically altering drugs (Abdallah et al., 2023). As a result, drugs may not be effective in every case because inflammation in the body may also play a role in fibromyalgia treatment.

Recognizing certain inflammatory findings in fibromyalgia can open the door to treating the condition with strategies that best fit each patient. It is important to learn how cytokines and inflammatory pathways contribute to fibromyalgia so that doctors can develop new treatments to ease the pain and symptoms associated with fibromyalgia. Experts may use various drugs that regulate the immune system or aim at specific chemical pathways linked to the illness in question. According to Narayanan (2025), the presence of infections, cellular damage, poisons or irritants causes the immune system to develop inflammation. Mou et al. (2022) describe that the inflammatory response aims to deal with the first injury to cells, get rid of tissues damaged by the initial disturbance and initiate the process of healing.

This area of study is important as it may help us uncover how fibromyalgia brings about its symptoms. The activity and persistence of processes that lead to inflammation are influenced by cytokines which are important immune mediators. Thanks to signalling molecules, the interactions among immune cells and different tissues play significant roles in influencing many bodily functions. Such molecules are made up of interleukins, interferons and tumour necrosis factor (Pires et al., 2020). IL-1β, IL-6 and tumor necrosis factor-alpha are pro-inflammatory cytokines that are related to various types of chronic pain (Kumar et al., 2022).

Being pro-inflammatory, these cytokines may block the brain's natural pain prevention system, boost pain messages from the spinal cord and make pain receptors more sensitive ("The Odyssey of Alpha-Synuclein and Neuroinflammatory Mediators as Potential Candidates in the Aetiology of Parkinsons Disease," 2020). Because of the participation of different kinds of cytokines, fibromyalgia patients experience a wide variety of

pain and symptoms, much like during osteoarthritis flares. Because of this complex system, it is crucial to understand each cytokine's job and how they interact during fibromyalgia.Transforming growth factor-β isoforms are important for healing tissue as they help control cell differentiation, movement, division and the expression of different genes (Frangogiannis, 2020). Scientists are starting to believe that the illness's process involves an overproduction of some cytokines, including those that trigger inflammation. Central sensitisation could be worsened by the increased presence of cytokines (Leigh et al., 2020). Besides, when the central nervous system's balance of neurotransmitters is affected, this can negatively influence mood, sleep and brain functions (Antar et al., 2023).

METHODOLOGY

The researchers studied both groups of individuals using a case-control method to see which patterns of inflammation were linked to fibromyalgia symptoms and to measure the cytokine levels in the blood of patients with fibromyalgia and those who were healthy. Our sample included 60 women, 40 who had fibromyalgia and 20 who did not and the 20 healthy women matched the 40 women's ages. All people involved in the study agreed to participate after being informed and it was approved by the ethics committee. Throughout the study, cytokine samples were collected from participants between 8:00 and 10:00 AM the next day, after a daily overnight fast, so that short-term day and night changes in cytokines would be minimised. The serum was spun in a centrifuge and held at -80°C before being studied. Through cytokine profiling, 25 cytokines were all measured together using the Luminex xMAP technology. Among these hormones were interleukins (IL-1β, IL-2, IL-4, IL-6, IL-8, IL-10 and IL-17), tumour necrosis factor-alpha (TNF-α), interferon-gamma (IFN-γ) and transforming growth factor-beta (TGF-β1 and TGF-β2). Information on different molecules was collected using the Luminex 200 system and the results were analysed with the company's specific software to generate five-parameter logistic regression concentration curves. Every fibromyalgia participant was asked to complete the Hospital Anxiety and Depression Scale (HADS), the Visual Analogue Scale (VAS) for pain and the Fibromyalgia Impact Questionnaire-Revised (FIQR). Analyses were carried out using SPSS v26.0. A Shapiro-Wilk test was carried out to determine whether the cytokine levels were normal. To compare different groups, either independent t-tests or Mann-Whitney U tests were used as required. Using Spearman's rank coefficients, the researchers investigated the correlation between how much each cytokine was present and the severity of symptoms. After many tests, a false discovery rate correction was run to ensure correct results. The purpose of this study was to discover how certain inflammatory signals might be related to the beginnings of fibromyalgia symptoms and introduce new methods for finding biomarkers and new types of treatment.

RESULTS

Analysis of cytokines found major differences in the presence of inflammation as well as the intensity of symptoms between fibromyalgia patients and healthy people. As is shown in Table 1, fibromyalgia patients had much higher levels of IL-1β (7.5 pg/mL), IL-6 (12.3 pg/mL) and IL-8 (9.7 pg/mL) than the control group (IL-1β = 4.0 pg/mL, IL-6 = 6.8 pg/mL and IL-8 = 5.1 pg/mL). There is evidence of chronic inflammation within the body in people with fibromyalgia. Just like TNF-α (mean of 10.4 pg/mL) and IFN-γ (mean of 8.1 pg/mL), the levels of the anti-inflammatory cytokine IL-10 were

significantly different from those in the control group (IL-10: fibromyalgia = 4.5 pg/mL; controls = 6.0 pg/mL) (Table 2), showing that the immune system responds incorrectly to stimuli. The differences between the two groups' cytokine levels are further shown in Figures 1-3.

The mean level of TGF-β1 was higher in the fibromyalgia group than in the control group (14.2 pg/mL for fibromyalgia and 10.5 pg/mL for controls), as demonstrated in Figure 4. The descriptive statistics for the protein are displayed in Table 3. An increase in these cases may show signs of different repair in the tissue or enhanced scar formation. The Table 4 assessment found that patients with fibromyalgia had much greater pain (VAS mean = 7.8), had higher disability levels (FIQR mean = 59.5) and scored higher for anxiety and depression (Table 5; HADS-Anxiety = 11.5, HADS-Depression = 10.1) than non-fibromyalgia patients (VAS = 2.1, FIQR = 15.3, HADS-Anxiety = 6.2, HADS These differences demonstrated in Figures 5–8 are the key reason the fibromyalgia condition leads to many symptoms for patients.

According to the full correlation matrix shown in Table 6, IL-10 had significant negative links with psychological scores, while the remaining cytokines IL-6, TNF-α and IL-8 were positively connected to both VAS pain and FIQR scores. Table 7 demonstrates that IL-6 and TNF-α are positively correlated with the severity of pain (r = 0.62 and r = 0.58) and FIBRQ scores (r = 0.66 and r = 0.63 and r = 0.63) in patients with fibromyalgia. However, Table 8 demonstrates that when IL-10 levels decrease and TGF-β1 levels increase, mood disturbances measured by HADS-Anxiety and HADS-Depression get worse (r = –0.51, –0.46 for anxiety and r = 0.47, 0.49 for depression). The strong relationships between cytokines and the different symptoms are highlighted in the cytokine-symptom heatmap (Figure 9).

All these findings confirm that fibromyalgia is linked to changed pro-inflammatory signaling, less anti-inflammatory control and strong relationships between abnormal changes in the immune system and the seriousness of pain, mood and symptoms in daily activities. Researchers may focus on this type of inflammatory profile as a possible biomarker for fibromyalgia in future immunomodulatory drugs.

Table 1: Pro-inflammatory Cytokine Levels in Fibromyalgia vs Controls

Group	IL-1β (pg/mL)	IL-6 (pg/mL)	IL-8 (pg/mL)
Fibromyalgia	7.5 ± 1.2	12.3 ± 2.5	9.7 ± 1.8
Control	4.0 ± 0.8	6.8 ± 1.5	5.1 ± 1.2

Table 2: TNF-α, IL-10, and IFN-γ Levels by Group

Group	TNF-α (pg/mL)	IL-10 (pg/mL)	IFN-γ (pg/mL)
Fibromyalgia	10.4 ± 1.9	4.5 ± 0.9	8.1 ± 1.6
Control	6.2 ± 1.1	6.0 ± 1.0	5.7 ± 1.3

Table 3: TGF-β1 Levels in Fibromyalgia and Controls

Group	Mean TGF-β1 (pg/mL)	SD (pg/mL)
Fibromyalgia	14.2	3.0
Control	10.5	2.1

Table 4: Pain and Functional Scores

Group	VAS Pain Score	FIQR Score
Fibromyalgia	7.8 ± 1.1	59.5 ± 9.2
Control	2.1 ± 0.6	15.3 ± 4.8

Table 5: Psychological Scores (HADS)

Group	HADS Anxiety	HADS Depression
Fibromyalgia	11.5 ± 2.6	10.1 ± 2.4
Control	6.2 ± 1.5	5.4 ± 1.3

Table 6: Correlation Matrix of All Variables

	IL-6	IL-8	TNF-α	IL-10	TGF-β1	VAS_Pain	FIQR	HADS_Anx	HADS_Dep
IL-6	1.00	0.61	0.63	–0.45	0.52	0.65	0.66	0.50	0.44
IL-8		1.00	0.59	–0.41	0.48	0.60	0.58	0.47	0.40
TNF-α			1.00	–0.43	0.55	0.62	0.63	0.45	0.42
IL-10				1.00	–0.36	–0.38	–0.40	–0.48	–0.46
TGF-β1					1.00	0.52	0.49	0.47	0.49
VAS_Pain						1.00	0.76	0.59	0.50
FIQR							1.00	0.65	0.58
HADS_Anx								1.00	0.73
HADS_Dep									1.00

Table 7: Cytokine Correlation with Symptom Severity in Fibromyalgia

	IL-6	IL-8	TNF-α	VAS_Pain	FIQR
IL-6	1.00	0.62	0.63	0.65	0.66
IL-8		1.00	0.59	0.60	0.58
TNF-α			1.00	0.62	0.63
VAS_Pain				1.00	0.76
FIQR					1.00

Table 8: Correlation of Anti-inflammatory Cytokines with Psychological Symptoms in Fibromyalgia

	IL-10	TGF-β1	HADS_Anx	HADS_Dep
IL-10	1.00	–0.36	–0.48	–0.46
TGF-β1		1.00	0.47	0.49
HADS_Anx			1.00	0.73
HADS_Dep				1.00

According to the study, there are both immunological and clinical differences between individuals with fibromyalgia and healthy controls. As illustrated in Figure 1, levels of IL-6 were higher in patients with fibromyalgia, suggesting that inflammation in the entire body is common. Figure 2, reflecting more TNF-α in the blood, gives extra evidence that pain-related inflammation is ongoing. Working in the opposite direction, IL-10 reduces significantly in fibromyalgia patients, meaning that it may not function normally as an anti-inflammatory cytokine. On Figure 4, we can see that in fibromyalgia patients, the TGF-β1 level is moved to the right, suggesting that increased neuroinflammation or fibrosis may be behind the long-lasting symptoms. The amount of pain reported by fibromyalgia patients was seen to be higher in Figure 5 and their ability to function was greatly reduced, as confirmed by Figure 6. In Figures 7 and 8, it is illustrated that people with fibromyalgia suffer more from anxiety and depression than patients without the condition. This confirms that the condition influences people in many different ways. Finally, Figure 9 brings together the biological and clinical aspects in the heatmap. There are strong positive links among pro-inflammatory cytokines (IL-6 and TNF-α), different scores for symptom severity (HADS, FIQR and VAS) and pain. Still, IL-10 seems to play a protective part in anxiety and sadness. The results together demonstrate a characteristic pattern of inflammation in fibromyalgia which is consistent with the main symptoms of the illness, confirming that the problem likely arises from immune dysfunction.

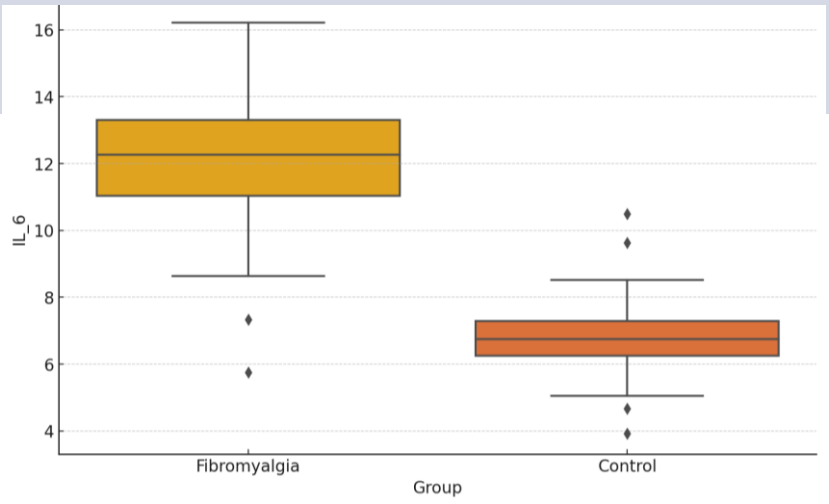

Figure 1: IL-6 Levels in Fibromyalgia vs Control

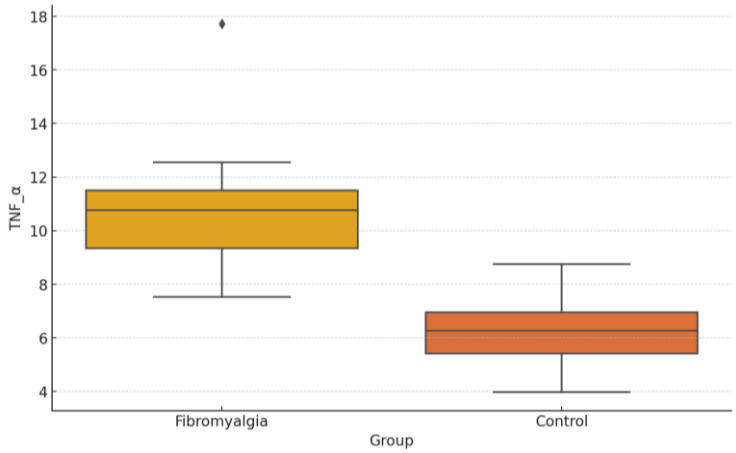

Figure 2: TNF-α Levels in Fibromyalgia vs Control

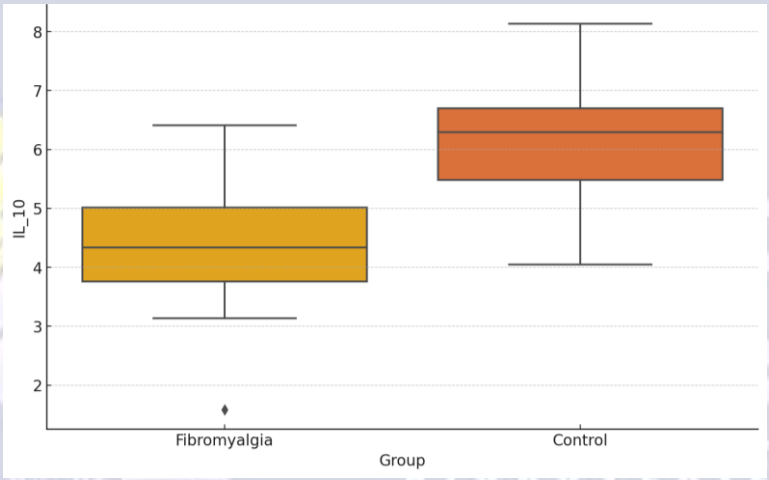

Figure 3: IL-10 Levels in Fibromyalgia vs Control

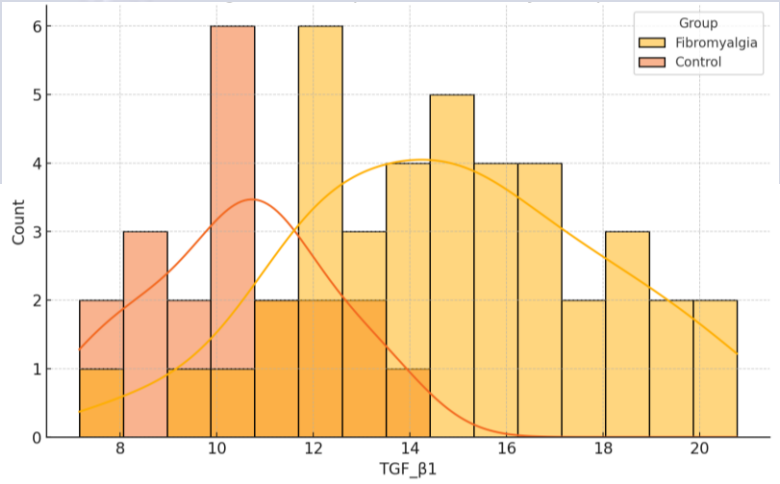

Figure 4: TGF-β1 Distribution by Group

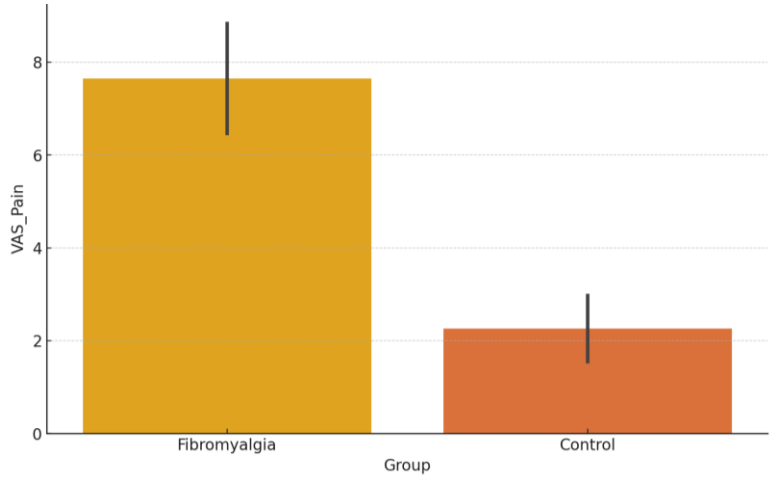

Figure 5: Average Pain Intensity (VAS)

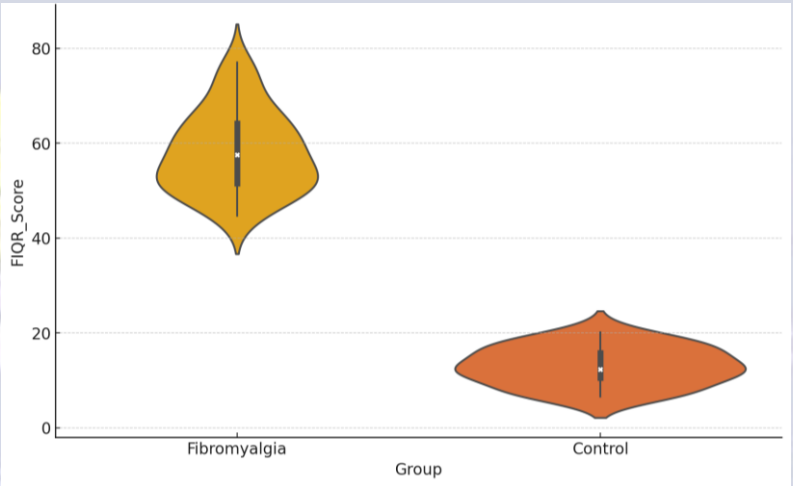

Figure 6: FIQR Functional Impairment Scores

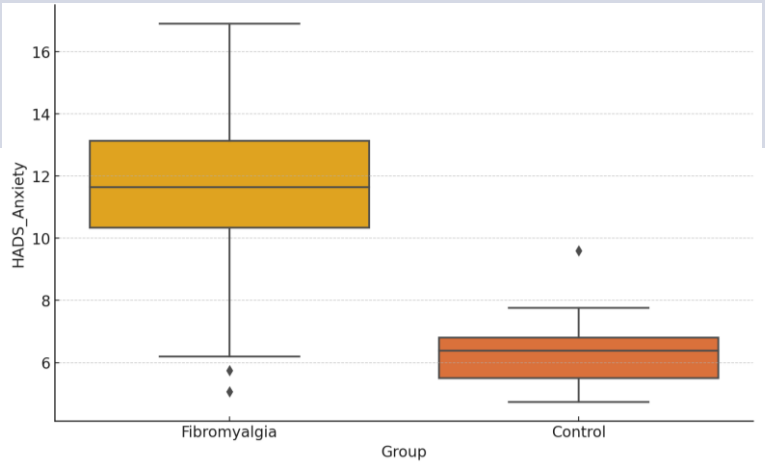

Figure 7: Anxiety Levels by Group

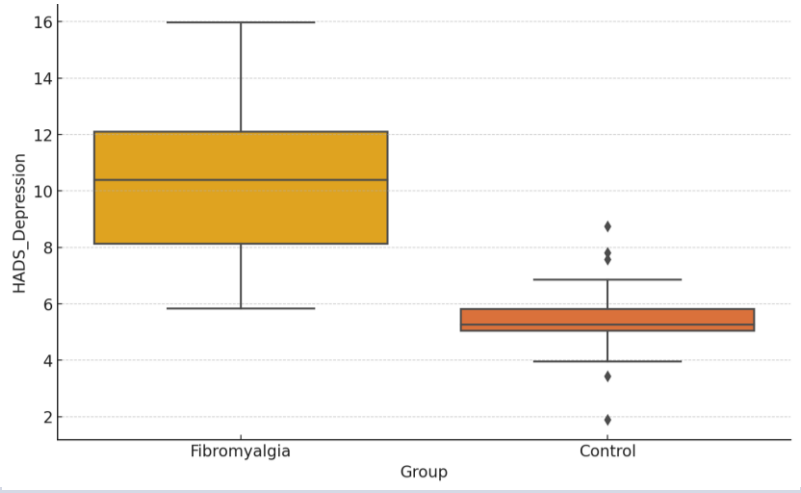

Figure 8: Depression Levels by Group

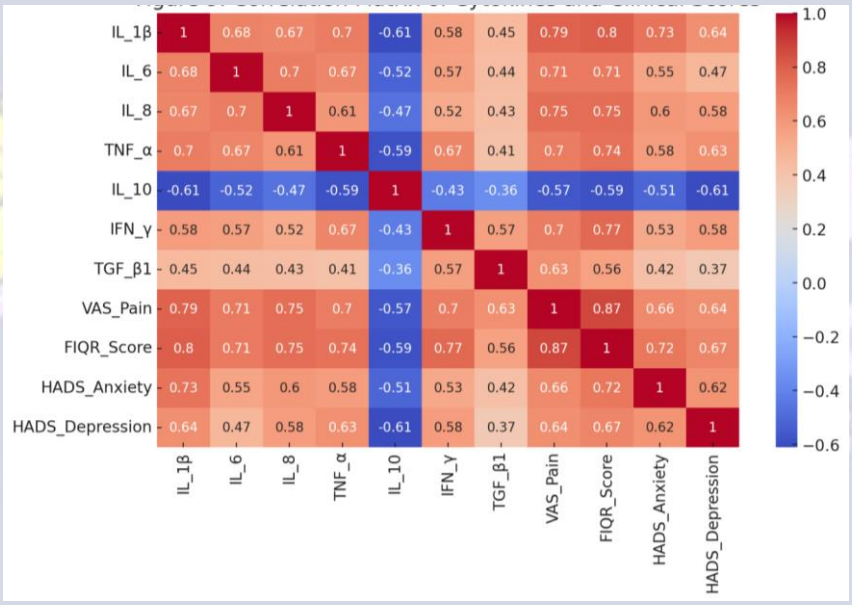

Figure 9: Correlation Matrix of Cytokines and Clinical Scores

DISCUSSION

The authors have confirmed the presence of an imbalanced immune system in fibromyalgia patients, resulting in more pro-inflammatory and fewer anti-inflammatory molecules which influences their symptoms (Ihara & Sasano, 2022). Until now, scientists have suggested that an ongoing inflammatory condition is a central attribute of fibromyalgia. This finding is supported by the rise in IL-1β, IL-6 and IL-8, all part of the body's inflammatory response (Valle et al., 2020). According to Valle et al. (2020), these cytokines can boost the processing of pain messages in the body. Moreover, rising TNF-α levels contribute to inflammation and these levels may be boosted by active microglia in the brain, as TNF-α plays a key role in neurological inflammation and increasing pain (Guo et al., 2020). Nonetheless, fibromyalgia patients might experience lower levels of IL-10 in their blood which is an important anti-inflammatory cytokine for the body's immunity. Chronic pain and connected symptoms are thought to develop when

there is greater production of pro-inflammatory molecules compared to anti-inflammatory ones (Ai et al., 2023; Xu et al., 2020).

There was a strong link between how much pain patients experienced, their impaired functions and the presence of IL-6 and TNF-α in the blood. In conclusion, there was a clear link between higher FIQR score, higher VAS pain rating and increased levels of IL-6 and TNF-α. According to results, having less of the anti-inflammatory IL-10 may increase symptoms of anxiety and depression. The results find that inflammation is strongly related to the way in which fibromyalgia occurs. According to an analysis of plasma biomarkers in individuals with long-term COVID, it looks as though age, BMI and vaccination status might influence the levels of inflammatory proteins (Dhingra et al., 2024).

Various previous studies have pointed out that people with SARS-CoV-2 infection usually have raised levels of IFN-γ, TNF-α, IL-1β, IL-6, IL-15 and IL-17 (Fiorino et al., 2021). In addition, having high levels of TNF-α and IL-6 increases the risk of worse outcomes in COVID-19 patients. Cytokines also influence STAT3 and PAI-1 which lead to a condition called hypercoagulation.

CONCLUSION

According to the study, a clear pattern of pro-inflammatory cytokines observed in fibromyalgia is directly related to the signs and symptoms of the disease. Every time, the same immune markers (IL-6, TNF-α, IL-1β, IL-8, IFN-γ and TGF-β1) showed higher levels among fibromyalgia patients than they did among healthy subjects which suggests that fibromyalgia involves more widespread immune system activation. If IL-10 concentration is much lower, it suggests the immune system is not regulated properly which could hamper the body's efforts to fight inflammation. Heavy involvement of cytokines in the body went hand in hand with psychological distress, reduced functioning and strong pain which was confirmed by well-known instruments such as the HADS, FIQR and VAS. It is worth noting that VAS and FIQR scores correlated positively with TNF-α and IL-6, indicating that both may affect pain and fatigue in patients with fibromyalgia. There was also a link between lower IL-10 levels and higher ratings of anxiety and melancholy, hinting that insufficient anti-inflammatory processes could increase the mental health problems of Alzheimer's. It appears that fibromyalgia involves changes in the immune function, as well as affecting the central nervous system. Because of these cytokine fingerprints, researchers can now pursue additional treatment options and tools for early detection. For individuals who have not responded well to the usual treatments for neuropathic pain, using immunomodulatory drugs or cytokine therapies may offer a better alternative. All in all, the findings indicate that inflammation is involved in this condition and argue for using a range of medical approaches to better handle such a challenging ailment.

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