RESEARCH ARTICLE

Alterations in the levels of cytokines associated with Th17 cells in the plasma of Guillain-Barre syndrome patients

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Abstract

Objective: To compare the cytokines associated with T helper 17 cells between Guillain-Barre syndrome patients and healthy controls.

Method: The cross-sectional, comparative study was conducted from March 2024 to February 2025 at the Department of Immunology, University of Health Sciences, Lahore, Pakistan, and comprised Guillain-Barre syndrome patients in group A and healthy controls in group B. Plasma concentrations of interleukin-6 (IL-6), interleukin-17, interleukin-21, interleukin-22 and interleukin-23 in both the groups were quantified using enzyme-linked immunosorbent assay. Data was analysed using SPSS 24.

Results: Of the 88 subjects, 44(50%) were in group A with mean age 41.6±11.8 years, while 44(50%) were in group B with mean age 39.3±12.4 years. Both the groups had 26(59%) male and 18(41%) female subjects. Serum interleukin-6, interleukin-17 and interleukin-22 levels were significantly higher in group A patients than group B controls ($p<0.05$). In contrast, reduced serum interleukin-23 levels were found in the group A compared to group B, but the difference was not significant ($p>0.05$). Correlation analysis among cytokine levels showed that serum interleukin-21 levels were correlated with interleukin-22 levels in group A patients. Interleukin-17 concentrations did not show any significant relationship with other cytokine levels ($p>0.05$). Interleukin-6 was negatively correlated with disease duration ($p<0.05$). No significant associations were found among cytokine levels, disease subtypes and severity ($p>0.05$).

Conclusion: The activity of T helper 17 cytokines was significantly elevated in Guillain-Barre syndrome patients, underlining the contribution of T helper 17 cell cytokines in Guillain-Barre syndrome and implying that these cytokines can be targeted for therapeutic modulation of the immune response.

Key Words: Guillain-Barré Syndrome, Th17, Cytokines, ELISA.

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Introduction

Guillain-Barré syndrome (GBS) is a disorder that affects the peripheral nervous system, and is triggered by an abnormal immune response that may result from infectious or non-infectious agents. Patients present with a sudden onset of flaccid weakening of the lower or upper limbs or both, which may progress to neurological impairment within four weeks. There are different subclasses of GBS based on electrophysiological features, including acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN), and acute inflammatory demyelinating polyradiculoneuropathy (AIDP).[1] The worldwide incidence of GBS is 1-2 in every 100,000 people per annum, and most patients are infected before the appearance of disease symptoms. GBS cases have been reported in Pakistan, but there is limited data available regarding the incidence of GBS in Pakistan. The overall GBS prevalence in the Pakistani population is 1.1/100,00/year to 1.8/100,000/year[2]. Preceding infections by microorganisms, such as Campylobacter jejuni, Epstein-Barr virus, dengue, Zika virus and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), have been reported in different studies.[3, 4]

GBS is thought to develop through various immunological reactions, including macrophage and complement system activation, cytotoxicity mediated by T cells, and demyelination and axonal damage to peripheral nerves. The combined effect of many cytokines, including macrophages, T lymphocytes and B lymphocytes, plays an important role in immune cell differentiation and activation. The role of cytokines as important mediators has been recognised in various upstream and downstream processes in several inflammatory disorders. Alterations in certain cytokines are involved in the disease onset and severity and severity.[5]

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Cytokines secreted by different T helper (Th) cells, including Th1, Th2 and Th17 cells, are thought to be involved in the onset and progression of GBS. Furthermore, cytokines are now commonly known as biomarkers that may predict treatment responses.[6] However, the exact immunological reactions that affect GBS risk and severity in most patients have not been clearly described. This knowledge gap can be addressed by investigating the roles of cytokines in GBS. Previous research revealed that the pathophysiology of GBS involves several cytokines, including tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), IL-17, IL-22, IL-27 and chemokine ligand 10 (CXCL10).[7-9] However, other studies have not reported any visible changes in the levels of these cytokines between GBS patients and controls.[10] Additionally, one study reported decreased levels of blood IL-17 in GBS patients.[11]

Although some studies have documented the role of Th17 cells and associated cytokines in GBS pathogenesis, data remains inconsistent.[12,13] To precisely recognise the association between Th17 cells and the pathology and risk of GBS, it is important to investigate the levels of priming cytokines (IL-6 and IL-23) and effector cytokines (IL-17, IL-21 and IL-22) of the Th17 pathway.

The current study was planned to compare the cytokines associated with Th17 cells between GBS patients and healthy controls.

Subjects and Methods

The cross-sectional, comparative study was conducted from March 2024 to February 2025 at the Department of Immunology, University of Health Sciences, Lahore, Pakistan. After approval from the institutional ethics review committee, the sample size was calculated using n= 〖(Z_(1-β)+Z_(1-α⁄2))〗^2 (σ_1^2+σ_2^2)/(μ1-μ2)2 formula with 90% power and 95% confidence interval (CI) with OpenEpi.[7,14] The sample was collected using convenience sampling technique. The study included patients aged 18–60 years diagnosed with GBS (Brighton diagnostic criteria) from the Punjab Institute of Neurology, The General Hospital, Lahore.[15] Equal numbers of age- and gender-matched healthy participants were subsequently enrolled. The patients were in group A, while the controls were in group B. Written informed consent was obtained from all the subjects. Those excluded were patients with malignant tumours, autoimmune and allergic disorders, chronic inflammatory demyelinating polyradiculoneuropathy, and those who had received hormone, plasmapheresis or intravenous immunoglobulin (IVIg) treatment. Individuals who had a neurological disease, systemic autoimmune disorder, or any infection within the preceding two months were also excluded.

For all the patients, clinical parameters, including age, gender, previous history of infections, time from onset to nadir, sensory disturbances, and cranial nerve deficits were collected.

Under aseptic conditions, approximately 3mL of peripheral blood was drawn via venipuncture and placed in gel vacutainers. Serum was separated from whole blood by centrifugation at 3500rpm for 10min. Serum samples were stored at -80°C after aliquoting. Cytokine levels were quantified using human IL-6, IL-17, IL-21, IL-22 and IL-23 enzyme-linked immunosorbent assay (ELISA) kits (Bioassay Technology Laboratory, Birmingham, United Kingdom). The absorbance values of the appropriate substrates were recorded at 450nm using an automated ELISA plate reader (iMark, Bio-Rad Laboratories, California, United States). The levels of these cytokines were determined by using standard curves.

Data was analysed using SPSS 24. Data was presented as median with interquartile range (IQR) for non-parametric variables or mean ± standard deviation for parametric variables. Independent sample t-test was used to examine the differences between quantitative variables when the data was normally distributed. Mann-Whitney U test was used to compare the median values of the variables that were not normally distributed. Correlations for parametric data or Spearman's correlation coefficients for non-parametric data were used to determine the relationships between different cytokines. Statistical significance was set at $p<0.05$.

Results

Of the 88 subjects, 44(50%) were in group A with mean age 41.6±11.8 years, while 44(50%) were in group B with mean age 39.3±12.4 years. Both the groups had 26(59%) male and 18(41%) female subjects. The most common GBS subclass was AMAN 21(47.7%), followed by AMSAN 15(34.1%), AIDP 7(15.9%) and Miller Fisher syndrome (MF) 1(2.3%). There were 24(54.5%) group A patients with antecedent infections. Gastrointestinal infections were the most common 19(43.2%). A higher proportion of group A patients 37(84.1%), had motor nerve damage, followed by 22(50%) with sensory damage (Table 1).

Serum IL-6, IL-17 and IL-22 levels were significantly higher in group A patients than group B controls ($p<0.05$). In contrast, reduced serum IL-23 levels were found in the group A compared to group B, but the difference was not significant ($p>0.05$) (Tabe 2).

A significant negative relationship was observed between

Table-1: Demographic and clinical data of Guillain-Barre syndrome (GBS) patients and healthy controls.

Variables		GBS patients (n=44)	Controls (n=44)	p- value
Age (Mean ± SD) years		41.6±11.8	39.3 ± 12.4	
Gender n (%) Male		26 (59%)	26 (59%)	1.00
Female		18 (41%)	18 (41%)	
CSF protein (mg/dl)		108 (57-358.2)	-	-
CSF WBCs/dl		2 (0-9)	-	-
Blood TLC x10^3/µL		12.75 (9-14.9)	-	-
GBS Subtype n (%)	MF	1 (2.3%)	-	-
	AIDP	7 (15.9%)	-	-
	AMSAN	15 (34.1%)	-	-
	AMAN	21 (47.7%)	-	-
Disease Severity* n (%)	Mild	17(39%)	-	-
	Severe	27 (61%)	-	-
Duration of weakness in days	10 (1-28)		-	-
Limb's involvement n (%)	Lower limbs	15 (34.1%)	-	-
	Both upper and lower	29 (65.9%)	-	-
Preceding Illness n (%)	No	20 (45.5%)	-	-
	GITinfection	19 (43.2%)	-	-
	Respiratory infection	5 (11.3%)	-	-
Respiratory involvement	No	34 (77%)	-	
	Yes	10 (23%)	-	
Nerve conduction studies n (%)	Sensory	7 (15.9%)	-	
	Motor	22 (50%)	-	
	*Both sensory and motor	15 (34.1%)	-	

n: Number, SD: Standard deviation, MF: Miller-fisher, AIDP: Acute inflammatory demyelinating polyradiculoneuropathy, AMAN: Acute motor axonal neuropathy, AMSAN: Acute motor and sensory axonal neuropathy, GIT: Gastrointestinal tract,
*patients had overlapping sensory and motor nerve involvement. *adapted from Hughes Functional Grading Scale criteria.

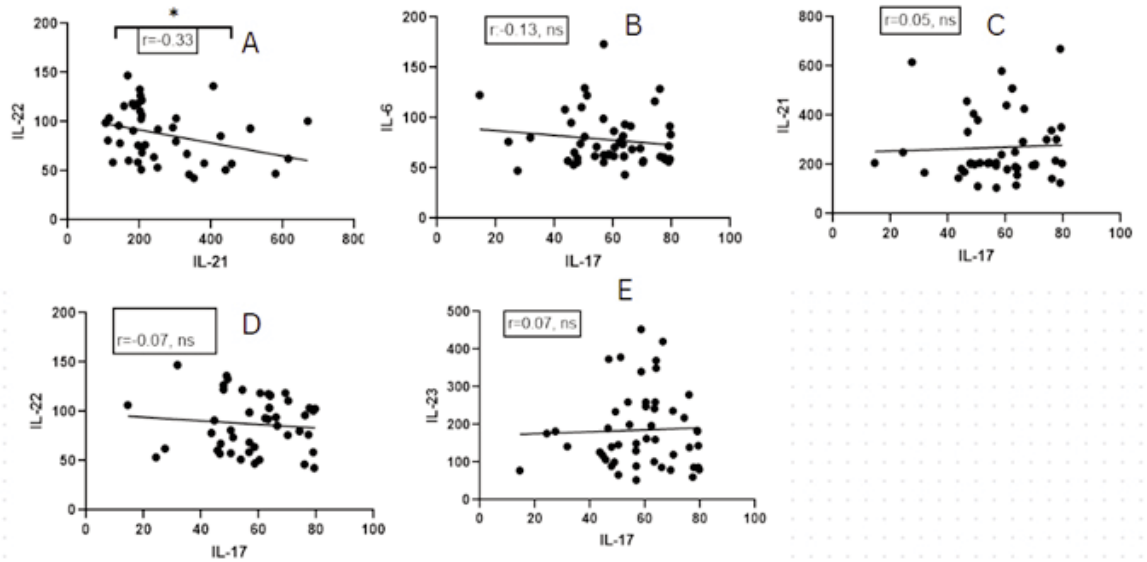

Figure-1: The correlation among different T helper 17-associated cytokine levels in Guillain-Barre syndrome GBS. (A) The correlation between the concentration of serum interleukin (IL)-21 and serum IL-22. (B) The relationship between serum IL-17 and serum IL-6 concentration. (C) The relationship between serum IL-17 and serum IL-21 concentration. (D) The relationship between serum IL-17 and serum IL-22 concentration. (E) The relationship between serum IL-17 and IL-23 levels in GBS.

Table-2: Cytokine concentrations associated with T helper 17 cells in Guillain-Barre syndrome (GBS) patients and healthy controls.

Cytokine	GBS Patients (n=44)	Healthy controls (n=44)	p Value
#IL-17A serum levels (ng/L)	57.3 ± 15.7	23.9 ± 14.6	<0.0001
IL-22 serum levels (ng/L)	87.3 ± 28.6	54.4 ± 18.3	<0.0001
*IL-6 serum levels (ng/L)	72.79 [31.61]	54.43[36.54]	0.003
IL-21 serum levels (ng/L)	206.5 [153.9]	184.1 [157.6]	0.08
IL-23 serum levels (ng/L)	161.44 [138.22]	192.1 [36.5]	0.39

#Mean±SD for parametric data. *Median [interquartile range] for non-parametric data. ng/L: Nanogram/literIL: Interleukin.

IL-22 and IL-23 concentrations among the disease grades (p=0.13, p=0.29, p=0.55, p=0.37 and p=0.21, respectively).

Discussion

It has been shown that cytokines and their interactions have a role in the pathophysiology of GBS.[16] In the current study, the diagnostic utility of Th17 cell cytokines (IL-6, IL-17, IL-21, IL-22 and IL-23) in patients with GBS and healthy controls was investigated and compared.

Th17 cells are the primary source of IL-17, also known as IL-17A, which directly activates the release of pro-

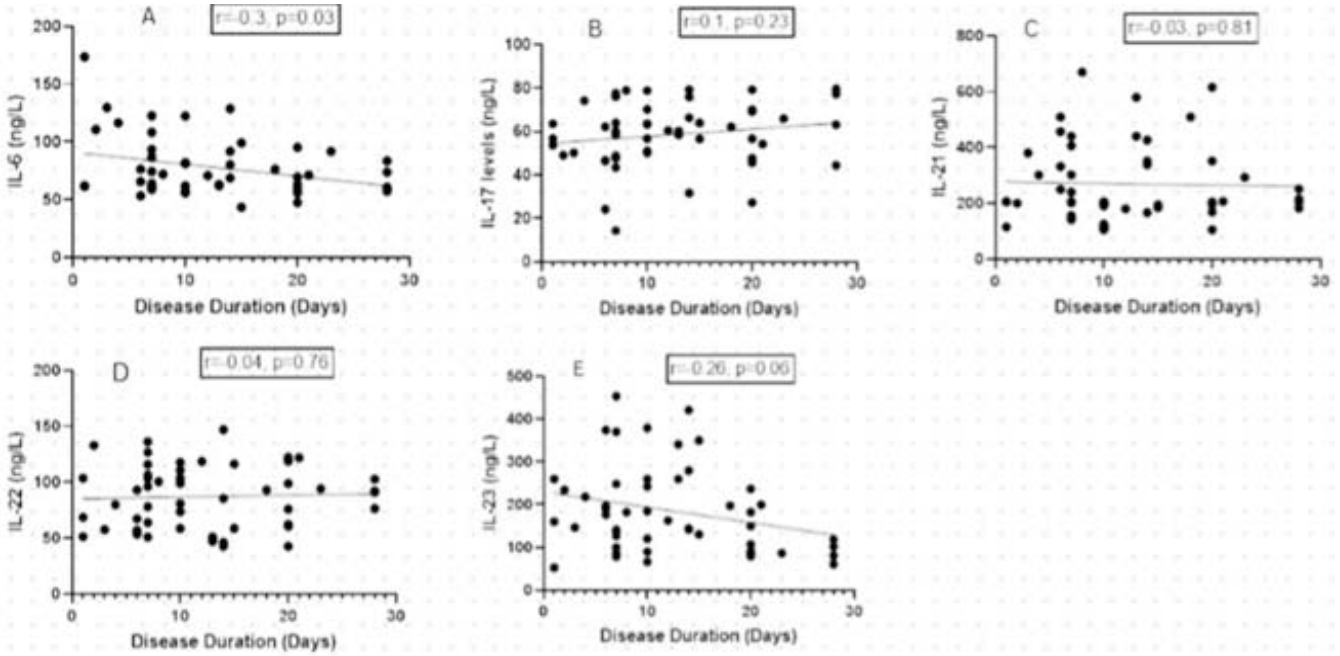

Figure-2: The correlation between different T helper 17-associated cytokine levels and duration of Guillain-Barre syndrome (GBS) onset. (A) The correlation between the concentration of serum IL-6 and disease duration. (B) The relationship between serum IL-17 concentration and GBS duration. (C) The relationship between serum IL-21 concentration and disease duration. (D) The relationship between serum IL-22 concentration and duration of disease. (E) The relationship between serum IL-23 and GBS disease duration.

IL-21 and IL-22 levels (r=-0.30, p=0.04, Figure 1A). Statistically non-significant correlations were found between serum IL-6 and IL-17 levels (r -0.13, p=0.52, Figure 1B). There was no significant relationship between IL-17 and IL-21 levels (r=0.05, p=0.71; Figure 1C). IL-17 did not affect IL-22 concentration (r=-0.07, p=0.64; Figure 1D) and IL-23 concentration (r=-0.07, p=0.65; Figure 1E).

There was no significant correlation between cytokine concentrations and disease duration (p>0.05), except for IL-6, which showed a significant negative correlation with disease duration (r=-0.3, p=0.03) (Figure 2). There were no significant differences in the levels of IL-6 (p=0.55), IL-17 (p=0.24), IL-21 (p=0.45), IL-22 (p=0.37) and IL-23 (p=0.54) among the different subtypes of GBS (p=0.72). No significant differences were observed in IL-6, IL-17, IL-21,

inflammatory cytokines and chemokines from fibroblasts, epithelial cells and endothelial cells.[17,18] The current study observed significantly elevated levels of IL-17 in GBS patients compared to those in healthy controls. Increased levels of IL-17 have also been observed in a Pakistani population with GBS.[19] According to previous studies, the elevated concentration of IL-17 in the plasma of GBS patients suggests that IL-17 may be important in the pathophysiology of GBS.[7,12] No significant correlation was detected between IL-17 levels and the clinical subgroups of patients in the current study, which was in line with literature.[13]

Increased levels of various pro-inflammatory cytokines have been documented in GBS patients.[20] The current

study also noticed that GBS patients had higher concentrations of inflammatory cytokines, such as IL-6, as reported by Wang et al. in 2015.[21] IL-6 may contribute to the pathogenesis of GBS hyponatraemia.[22] Th current findings are contradictory to those by Dahle et al. (2003) and Nicknafs et al. (2021), who found no significant differences in IL-6 concentrations between their patient and control groups.[10, 23] The patients in the current study were in the acute phase, providing the best explanation for this discrepancy. In the current study population, there was no significant association between the levels of IL-6 and other cytokines and the clinical subtypes of the patients.

IL-21 is involved in inflammatory processes and autoimmune diseases by stimulating Th17 lymphocyte production. In lupus erythematosus, an autoimmune disease, elevated IL-21 levels in both skin and blood have been documented.[24] IL-22 is commonly associated with epithelial and mucosal immunity, but its role in inflammation could enhance the demyelination process in GBS by activating immune cells that attack the myelin sheet or contribute to increased inflammation in the peripheral nervous system. Compared to healthy controls, the current GBS patients had significantly higher concentrations of IL-22 and elevated levels of IL-21. In line with previous studies[7,25], IL-22 levels were remarkably higher in GBS patients. In the current study, there was a significant correlation between IL-21 and IL-22 levels. There was no difference in the concentrations of IL-21 and IL-22, with disease severity comparable to that reported in a study conducted in Chinese population.[26]

IL-23 is thought to act as a pro-inflammatory cytokine, particularly during the initial stages of GBS. The current study observed that serum IL-23 concentrations in GBS patients were lower than those in healthy controls, but the difference was not significant. Nevertheless, in patients with GBS, there was no correlation between IL-23 and other cytokine levels. Doncel et al. also reported reduced IL-23 levels in GBS patients compared to healthy participants.[11] Contrarily, Peng et al. and Debnath et al. found that the serum levels of IL-23 in GBS patients were elevated during the acute phase of the disease compared to those in the control group.[16, 27]

This was a single-centre study, and the analysis was restricted to a limited panel of Th17-related cytokines without exploring other immune pathways or underlying mechanisms. Measurements were taken only during the acute phase, potentially overlooking variations in disease stages. Future studies should adopt longitudinal, multicentre designs with larger and more diverse cohorts, incorporate a broader range of immunological markers, and include functional and mechanistic analyses to better elucidate the role of cytokines in disease progression and therapeutic responses.

Conclusion

The elevated IL-6, IL-17 and IL-22 serum levels in GBS patients may suggest underlying Th17-mediated inflammatory response. Meanwhile, the findings of correlational analyses among these cytokines in GBS suggested that the interactions between IL-21 and IL-22 were potentially related to GBS pathogenesis.

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Conflict of Interest: None.

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AUTHORS' CONTRIBUTIONS:

BM & AI: Data acquisition, analysis, interpretation, drafting, final approval and agreement to be accountable for all aspects of the work.
SM: Data acquisition, interpretation, revision, final approval and agreement to be accountable for all aspects of the work.
RT: Concept, design, data interpretation, revision, final approval and agreement to be accountable for all aspects of the work.