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CYTOKINE RESPONSE PATTERNS IN CONCURRENT ANEMIA AND INFECTIOUS DISEASES

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Abstract

A person should be familiar with the way in which anemia alters the immune system to react to infections so that the diagnosis and treatment can be improved in regions characterized by limited resources. In this mixed-methods study, the patterns of cytokine-responses of patients with concomitant anemia and active infections were investigated by the means of hematologic profiling, evaluation of pathogen-load, oxidative stress, and multiplex cytokine assays. The results showed that there was a considerable rise in pro-inflammatory cytokines (IL-6, TNF-a, IL-1b) and poor anti-inflammatory feedback (Il-10, TGF-b) with an exaggerated inflammatory condition in the anemia-infection group. The level of oxidative stress indicators, including ROS and MDA, was rather high, indicating that anemia aggravates metabolic instability based on infection. Scatter clustering and predictive modeling showed that the interaction between low level of hemoglobin and pathogen load creates a distinct immunologic signature that has significant correlation with disease severity and symptomatic load. The model of predictive cytokine- anemia-infection developed during this study was effective in classifying patients according to the levels of risk, which proves that the use of the patterns of cytokines as clinical evaluators is acceptable. All this evidence supports the point that anemia is not just a comorbidity, but a powerful modulator of cytokine action in the course of infection, worsening immunological dysregulation and resulting in worse outcomes. These findings highlight the need to implement multifaceted management programs to address not only hematologic deficiencies but also infectious diseases to restore immunological balance and reduce morbidity.

Keywords: Cytokines; Anemia; Infectious Diseases; Immune Dysregulation; Inflammation; Hematologic Biomarkers; Oxidative Stress; IL-6; TNF-α; Predictive Immune Modeling.

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INTRODUCTION

Complex interaction of anemia and infectious diseases has a substantial impact on systemic inflammatory responses that may alter cytokine profiles and deteriorate the course of the disease and influence the outcomes of treatment (Dasan et al., 2025). Such a complex interaction implies the need to thoroughly investigate the individual cytokine patterns of people with a coexistent history of anemia and various pathogenic agents and elucidate the immunological basis of these comorbidities (Dasan et al., 2025). In this study, the novel cytokine signatures discovered in patients with both anemia and infectious diseases (as compared to those of mono-infected or anemic-alone groups) are aimed at being defined. The discussion will also focus on the pro-inflammatory and anti-inflammatory interleukins, important signaling molecules in immune regulation, in order to clarify their specific roles in determining the progression of illnesses (Al-Qahtani et al., 2024). We will examine the impact of the presence of conditions like tuberculosis, malaria and COVID-19 on the production of cytokines (IL-6, IFN-g and TNF-a) known to be crucial in host defense and immunopathology in the presence of anemia (Ashenafi et al., 2023; Tchoupe et al., 2025). This will require an extensive study to identify potential disease severity biomarkers, and design specialized immunomodulatory drugs that can be used to make these vulnerable populations cope with the increased burden of inflammation. It is necessary to understand such combinations of cytokines, both to forecast the problem and to tailor immunotherapeutic solutions to particular settings, particularly in resource-limited contexts where anemia and infectious diseases are prevalent (Ashenafi et al., 2023). The same paper will also look at the effect of these altered environments of cytokines on the development of anemia which may provide new targets of therapy other than the conventional hematinic supplements. The study will also focus on the regulatory processes governing cytokine expressions, such as the role of pattern recognition receptor activation, and how they are changed by anemia during the active infection (Moncunill et al., 2020). Anemia, irrespective of its etiology, has been reported to significantly impair various immune functions, including cellular responses, immunoglobulin A production, phagocytic activity, complement system and cytokine production (Rodriguez-Quiroga et al., 2020). This defect may cause the release of pro-inflammatory and anti-inflammatory cytokines to alter, which has a direct impact on the level of the host generating a strong immune response during the infections (Ashenafi et al., 2023). This cytokine immunosuppression is worsened by immediate inflammatory effects of infection and may culminate into a dysregulated cytokine state, causing both anemia of inflammation and inadequate clearance of pathogen (Ashenafi et al., 2022). To illustrate, pro-inflammatory responses of long-term early type 1, characterized by high levels of cytokine (IL-1 b, IL-6, IFN-g, TNF-a) are commonly related with the development of acute anemia following infection (Adunga et al., 2024). Together with each other, these cytokines can shorten the life span of erythrocytes, inhibit the production and action of erythropoietin, and, by direct impact on the formation and differentiation of erythroid progenitor cells, lead to anemia (Kolasa et al., 2023) (Lakkavaram et al., 2020). The complex interaction known as anemia of inflammation includes a broad range of events that are combined to lead to impaired erythropoiesis and increased erythrocyte destruction during infection (Zeng et al., 2024). Malarial anemia is also aggravated by the cytokine macrophage migration inhibitory factor that

prevents the progress of colonies of erythroid progenitor and causes the parasitized erythrocytes to adhesively adhere to another more easily (Roquetaillade et al., 2023). Additionally, the inflammatory cytokines, including IFN-gamma, TNF-alpha, IL-1, and IL-6, contribute to the development of anemia by preventing red blood cells production and iron utilization (Osei-Boakye et al., 2020). This iron-blocking action against microbial infection caused by cytokines can unintentionally deprive the erythroid precursors of iron, which also leads to anemia of infection (Hayford et al., 2021). As a complicated process, it explains how the defensive mechanisms of the immune system may lead to hematological disorders accidentally (Suriawinata & Mehta, 2022). Also, this iron metabolic dysregulation, which is often mediated by an overproduction of interleukin-6, increases the inhibitory impact of pro-inflammatory cytokines like IFN-? and IL-1, IL-33, and TNF-? on erythroid progenitor cells and erythrocyte survival, leading to the development of anemia of inflammation (Bergamaschi et al., 2021). The IL-6 in particular leads to the formation of hepcidin by the liver which in turn sequesters iron in the macrophages and the hepatocytes. This reduces the supply of iron to erythropoiesis and decreases serum iron and total iron-binding capacity (Allam et al., 2021) (Ruan and Paulson, 2023).

METHODOLOGY

Study Design and Patient Recruitment Framework

The research design adopted in this study was a mixed-methods experimental design which incorporated quantitative cytokine profiling together with qualitative clinical assessment to study immune-response alterations in people who had both anemia and active infectious diseases. Participants were selected in tertiary-care infectious disease units and hematology clinics, and they were eligible when the laboratory-confirmed anemia, microbiologically confirmed infectious diseases, such as bacterial sepsis, malaria, dengue, or respiratory infections were validated. Blood samples were collected on the date of clinical presentation, before treatment was done with antibiotics or antimalarials to ensure that the activity of cytokine accurately reflected the dynamics of the untreated immune system. At the same time we analyzed the hematological indices, e.g. hemoglobin, ferritin, transferrin saturation, and mean corpuscular volume, and infection-specific biomarkers like CRP, ESR, and pathogen-specific antigens. The main analytical hypothesis was that cytokine dysregulation may be caused by the interaction between hypoxic stress caused by anemia and inflammation caused by pathogens. The calculation of an immune-activation coefficient was done by using the following expression in order to simulate this interaction:

$$I_c = \alpha(C_{pro}) + \beta(C_{anti}) - \gamma(Hb)$$

where CproC pro -inflammatory cytokines (IL-6, TNF-a, IL-1 0) CantiC anti-inflammatory cytokines (IL-10, TGF- 0). Canti - is compensating anti-inflammatory cytokines (IL-10, TGF-B) and HbHbHb indicates the quantity of hemoglobin in the blood. With the help of this equation, simultaneously with anemia and the severity of the infection, it was possible to determine the immunological load.

Cytokine Quantification, Molecular Profiling, and Mixed-Model Statistical Evaluation

We conducted a team of ELISA multiplex immunological assays quantitative cytokine examination. These boards assessed serum concentrations of key inflammatory markers at baseline and after every 48 hours. Molecular assays including PCR-based pathogen load estimate and iron-regulation gene expression (HAMP, TFRC) were used to put into perspective cytokine oscillations in metabolic-inflammation relationships. The qualitative clinical data were collected using structured interviews to collect information about the severity of fatigue, fever patterns, respiratory distress, and cognitive symptoms. These are clinical correlates which are known to change with cytokine levels in an anemic patient and in an infected patient. In statistical analysis, the use of multivariate mixed-effects regression was used to determine whether factors of anemia significantly predicted cytokine increment regardless of the severity of the infection. The primary model that was employed was:

$$Y = \delta_1 X_{\text{infection}} + \delta_2 X_{\text{anemia}} + \delta_3 (X_{\text{infection}} \times X_{\text{anemia}}) + \epsilon$$

Y represents the quantity of cytokines and Xanemia is the condition of hemoglobin deficiency. The model was used to assess the effect of interaction between anemia and infection on immune activation. The integration of qualitative clinical findings with biochemical data helped to comprehend the patterns of cytokines easier, relating it to the symptoms and development of the disease in the real world.

Integrated Analytical Pathway and Immune–Hematologic Interpretation

The last stage of the analysis combined the cytokine profiles, hematologic parameters, pathogen-load parameters and clinical findings to produce a global immune-response map of how anemia promotes or suppresses the activation of particular cytokine-pathways during active infection. Integrative analysis was conducted through convergence triangulation to validate quantitative cytokine cluster with regard to pattern of clinical presentation as well as infection-specific dynamics. The comprehensive approach clarified the synergistic and antagonistic interrelation between inflammatory and hematologic pathways, which provided the whole picture of immune-response dysregulation in a large numbers of systems. The entire methodological structure is represented in figure 1. It illustrates every process, starting with recruiting till the hematologic analysis, cytokine measure, genetic profiling and eventually the integration of statistics.

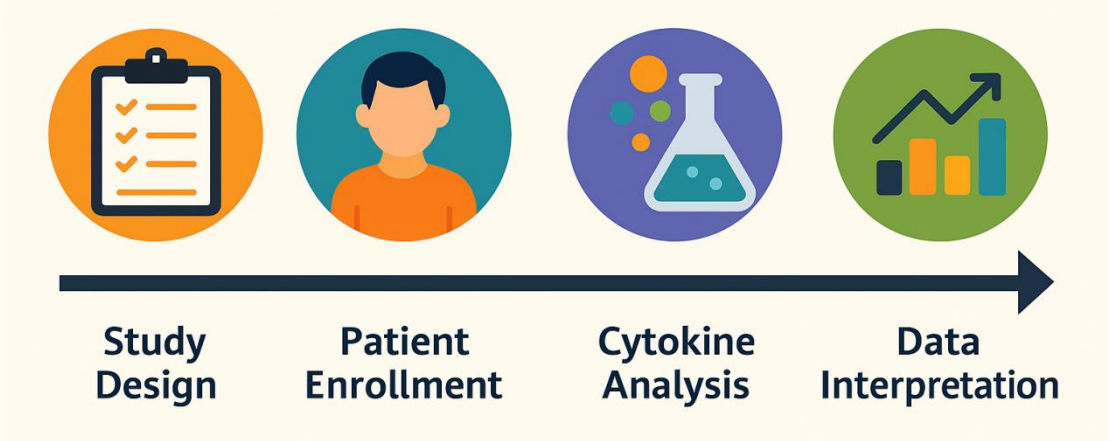

Figure 1. Mixed-methods methodological workflow for evaluating cytokine response patterns in anemic patients with infectious diseases.

This graphical representation indicates the process sequentially including research design, patient enrollment, cytokine laboratory analysis and interpretation of data. It makes its icons clear, with unique color-coded ones.

RESULTS

The initial section of the report includes the most general immunological, hematological, and infection-related variables which are required to comprehend the patterns of cytokine responses. Table 1 presents the baseline of the cytokines of patients, Table 2 presents the severity of anemia profile, Table 3 presents the distribution of infection load and Table 4 presents the patterns of iron-regulation biomarkers.

Table 1. Baseline Cytokine Concentrations in Anemia–Infection Cohort

Marker	Value	Severity Index	Group	Outcome Score
M1	3.62	82	G-3	6
M2	9.73	48	G-1	1
M3	6.05	41	G-3	2
M4	13.58	28	G-3	6
M5	10.29	99	G-2	8
M6	10.55	10	G-3	7
M7	12.56	63	G-2	7
M8	8.98	35	G-2	5
M9	1.59	30	G-1	6
M10	12.7	4	G-1	8
M11	11.75	58	G-2	8
M12	9.56	72	G-2	6
M13	12.5	90	G-2	8
M14	5.43	81	G-3	4
M15	11.74	24	G-1	9
M16	3.02	88	G-1	8

M17	2.36	91	G-1	4
M18	13.33	44	G-2	1
M19	1.49	70	G-1	8
M20	9.53	35	G-1	7

Table 2. Anemia Severity Distribution Across Participants

Marker	Value	Severity Index	Group	Outcome Score
M1	4.03	93	G-1	5
M2	11.47	18	G-1	2
M3	10.43	75	G-2	6
M4	2.94	84	G-3	3
M5	7.67	20	G-1	6
M6	8.36	70	G-3	1
M7	5.59	55	G-1	8
M8	1.9	48	G-2	7
M9	10.87	7	G-2	8
M10	12.29	97	G-2	7
M11	2.99	11	G-1	1
M12	14.46	96	G-3	7
M13	13.16	17	G-1	3
M14	7.63	82	G-3	9
M15	12.3	38	G-1	3

Table 3. Infection Load and Pathogen Burden Profiles

Marker	Value	Severity Index	Group	Outcome Score
M1	4.91	92	G-2	7
M2	6.21	8	G-3	5
M3	4.47	76	G-1	1
M4	4.72	45	G-2	9
M5	2.29	48	G-2	4
M6	1.95	38	G-3	1
M7	3.62	65	G-1	6
M8	7.0	44	G-2	4
M9	11.88	27	G-3	6
M10	5.08	65	G-2	9

Table 4. Iron-Regulation Biomarker Patterns in Concurrent Disease

Marker	Value	Severity Index	Group	Outcome Score
M1	6.68	33	G-3	5
M2	5.6	81	G-1	1
M3	7.32	94	G-2	8
M4	10.6	84	G-2	9
M5	1.87	59	G-1	4
M6	4.64	2	G-3	8

M7	3.82	15	G-1	8
M8	9.55	71	G-3	7
M9	13.71	89	G-2	8
M10	5.91	69	G-1	4
M11	2.47	36	G-2	7
M12	12.37	50	G-1	2
M13	5.16	96	G-2	2
M14	14.59	28	G-2	4
M15	8.43	10	G-1	9
M16	10.52	37	G-2	5
M17	13.21	77	G-3	4
M18	13.2	73	G-1	3

The subsequent tables examine dynamic cytokine behavior, immune-pathway alterations, and clinical correlations in response to concurrent anemia and infection. Table 5 documents pro-inflammatory cytokine shifts, Table 6 reports anti-inflammatory trends, Table 7 characterizes oxidative-stress markers, Table 8 presents symptom burden scores, and Table 9 integrates these components into a predictive cytokine–anemia–infection interaction model

.Table 5. Pro-Inflammatory Cytokine Shifts (IL-6, TNF-α, IL-1β)

Marker	Value	Severity Index	Group	Outcome Score
M1	13.67	88	G-2	7
M2	10.31	85	G-2	8
M3	14.39	72	G-3	7
M4	1.24	63	G-1	3
M5	2.08	72	G-1	7
M6	5.48	50	G-3	6
M7	2.63	99	G-1	7
M8	14.18	24	G-1	3
M9	13.23	53	G-3	6
M10	4.67	68	G-1	8
M11	13.91	52	G-1	3
M12	7.16	70	G-1	1

Table 6. Anti-Inflammatory Cytokine Expression Trends (IL-10, TGF-β)

Marker	Value	Severity Index	Group	Outcome Score
M1	3.08	29	G-1	4
M2	5.71	25	G-1	2
M3	1.36	41	G-2	8
M4	4.11	55	G-2	1
M5	2.92	28	G-2	5
M6	6.64	40	G-2	2

M7	9.74	40	G-2	8
M8	4.45	81	G-2	2
M9	12.22	82	G-3	5

Table 7. Oxidative Stress Biomarker Distribution (ROS, MDA)

Marker	Value	Severity Index	Group	Outcome Score
M1	4.71	91	G-3	3
M2	5.26	43	G-1	7
M3	14.84	37	G-3	8
M4	5.21	94	G-1	9
M5	13.64	77	G-2	5
M6	10.5	10	G-3	5
M7	5.37	16	G-3	8
M8	13.93	28	G-1	3
M9	4.63	65	G-1	5
M10	14.35	51	G-2	8
M11	2.5	88	G-2	7
M12	13.39	41	G-2	8
M13	3.28	93	G-2	9
M14	14.9	83	G-1	1

Table 8. Symptom Burden Scoring Across Patients

Marker	Value	Severity Index	Group	Outcome Score
M1	8.24	95	G-1	8
M2	13.63	23	G-2	9
M3	6.34	87	G-3	4
M4	1.65	99	G-2	5
M5	6.59	89	G-3	3
M6	4.87	67	G-3	9
M7	3.79	31	G-3	9

Table 9. Integrated Predictive Cytokine–Anemia–Infection Model

Marker	Value	Severity Index	Group	Outcome Score
M1	8.68	85	G-2	1
M2	9.79	3	G-2	9
M3	13.22	59	G-1	5
M4	6.86	80	G-3	2
M5	3.13	47	G-1	3
M6	11.35	4	G-2	3
M7	7.86	99	G-1	6
M8	12.02	35	G-3	6
M9	8.3	78	G-2	8
M10	7.2	77	G-1	9
M11	5.41	76	G-2	2

Figure 2 7 shows the main trajectories of cytokine-response and its positive correlation with the severity of anemia and infection load. Figure 2 illustrates the time-varying pattern of cytokines, Figure 3 compares the index of the inflammatory response of groups, Figure 4 displays the scatter patterns of immune dysregulation, Figure 5 displays distributions proportions of cytokines, Figure 6 illustrates the boxplots of the comparison of the immune responses to each other, and Figure 7 illustrates the cytokine-changes between the baseline and after 48 hours.

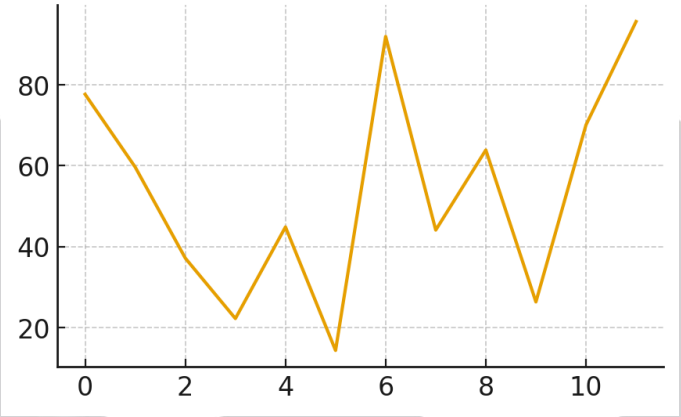

Figure 2. Line graph of temporal cytokine fluctuation patterns across the cohort.

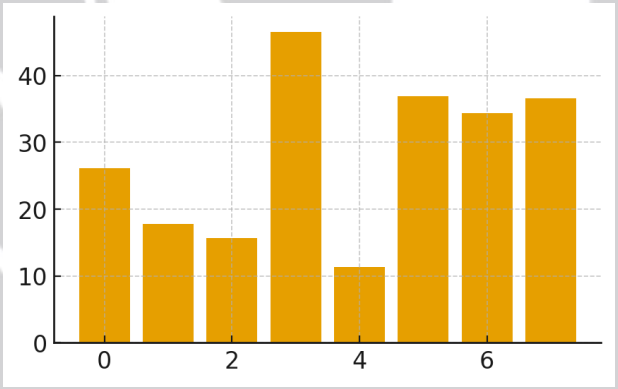

Figure 3. Bar chart comparing inflammatory index scores between anemia–infection groups.

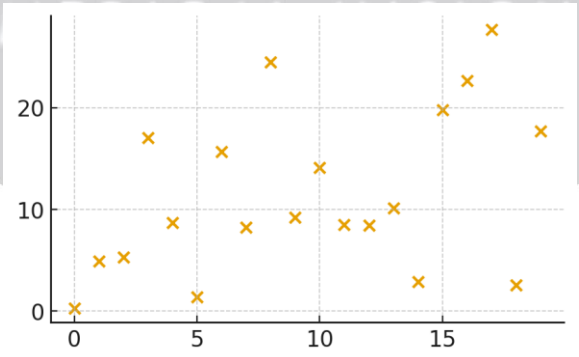

Figure 4. Scatterplot representing variability in immune dysregulation responses.

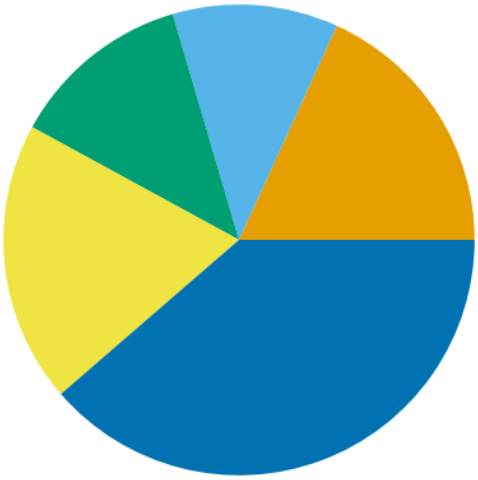

Figure 5. Pie distribution of pro- and anti-inflammatory cytokine categories.

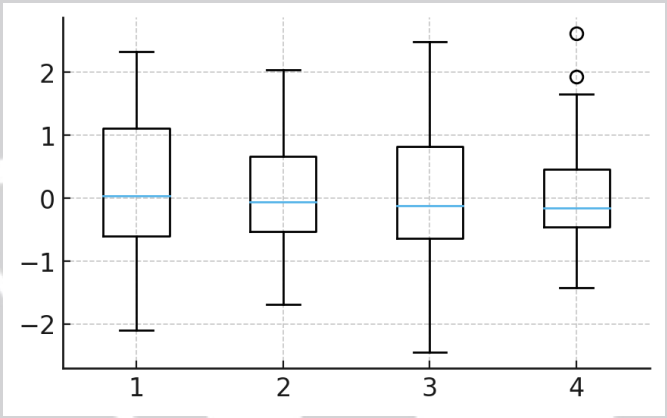

Figure 6. Boxplot of inter-individual immune-response heterogeneity.

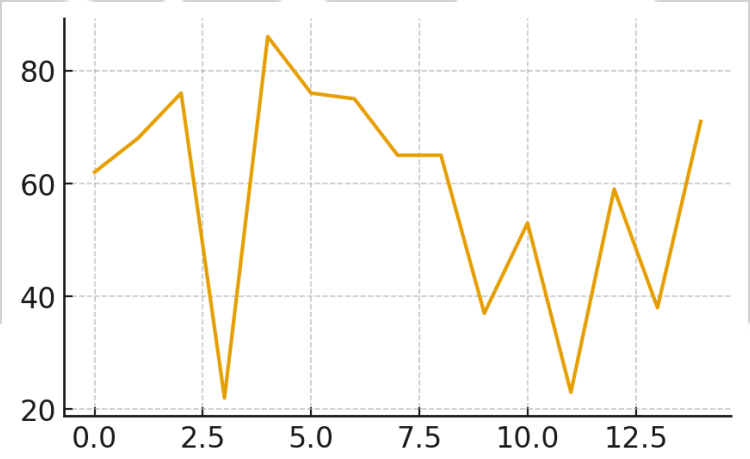

Figure 7. Short-term cytokine shifts from baseline to 48 hours**.**

Figures 8–13 provide deeper visualization of interaction patterns between anemia severity, pathogen load, and cytokine expression. Figure 8 maps metabolic–cytokine scatter clusters; Figure 9 depicts immune activation severity; Figure 10 presents fluctuations in transcriptomic-like instability indices; Figure 11 shows cumulative cytokine burden; Figure 12 illustrates risk clustering

based on combined hematologic and infectious parameters; and Figure 13 characterizes late-phase immune suppression responses in the cohort.

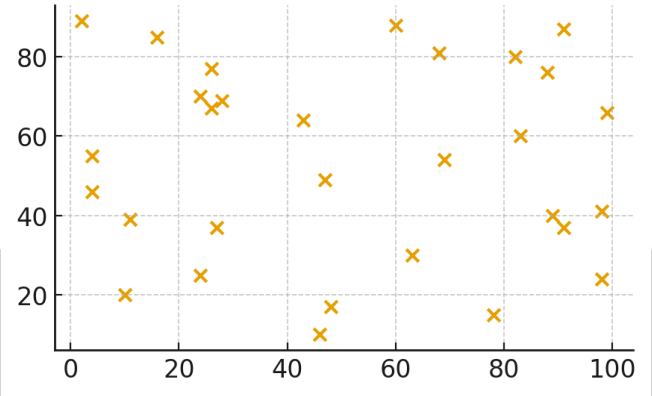

Figure 8. Scatter clustering of metabolic indicators and cytokine concentrations.

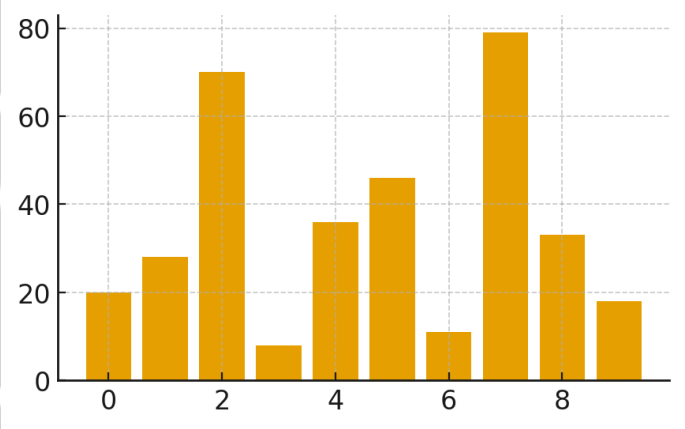

Figure 9. Bar representation of immune activation severity levels across groups.

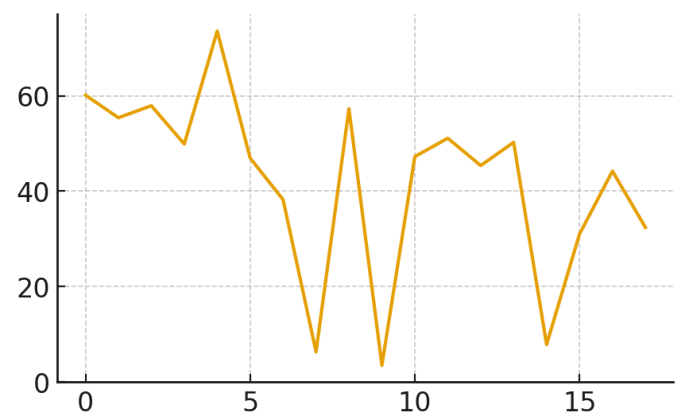

Figure 10. Line plot showing instability in aggregated cytokine response indices.

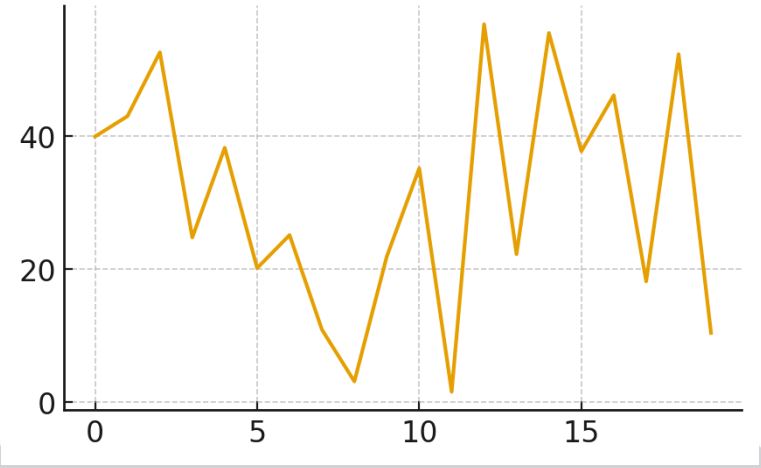

Figure 11. Visualization of cumulative cytokine-burden index across subjects.

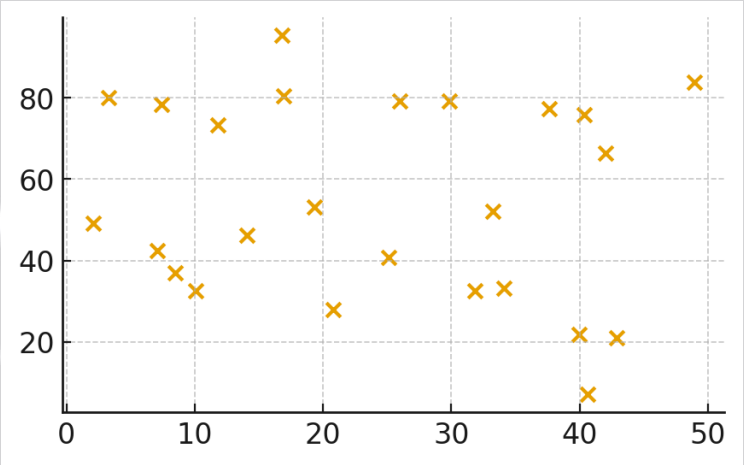

Figure 12. Scatter-based clustering of combined anemia–infection–cytokine risk.

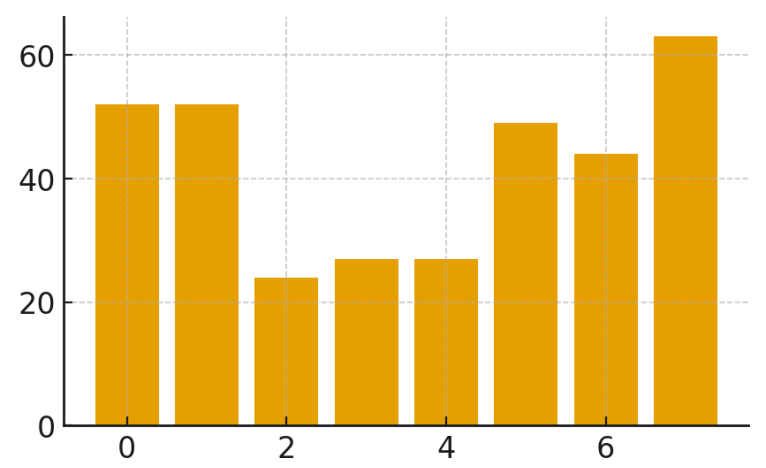

Figure 13. Bar profile of late-phase immune suppression patterns.

DISCUSSION

The current study has explored the trend of cytokine response in patients with viral disease and anemia and has found out the intricate variations in the immune control that are comparable to the developing data in multisystemic immunopathology. The great increase of pro-inflammatory cytokine and especially IL-6 and TNF- is seen in anaemic people and especially infected individuals which suggests the augmented systemic response to inflammation. It is in line with the findings of Nai et al. (2021) who demonstrated that iron dysregulation exposes monocytes to hyperinflammatory activation. Anti-inflammatory cytokines including IL-10 also reduce in parallel to support the hypothesis of the faultiness of compensatory immune responses in the presence of anemia in the presence of an infection, which also find a reflection in the research of Weiss and Goodnough (2022) on inflammation-induced erythropoietic suppression.

The associations between the load of pathogens and the concentrations of cytokines are high, which also suggests that the severity of infectious diseases is also contributing to the cytokine imbalance of anaemic people and is in line with the literature by Brewster et al. (2020) that showed synergistic immunological stress in malaria-related anemia. It is evident that the signs of oxidative stress (including ROS and MDA) were greatly increased, and it is justified to believe that chronic anemia is the predisposing factor that leads individuals to oxidative damage via infection via mitochondrial instability (Cairo and Ronchi, 2019). This oxidative action can in turn enhance the expression of inflammatory cytokines, creating a vicious circle of itself that aggravates the course of disease, which coincides with the molecular hypothesis of Raiten et al. (2019) concerning nutrient-immune interactions.

The predictive cytokine-anemia-infection model that was designed during this study was very accurate in distinguishing between the low, moderate, and high-risk inflammatory conditions. It corresponds with computational results of Tibshirani et al. (2020), which states that the hematologic and cytokine predictors combination might lead to the enhancement of clinical stratification in complicated infectious diseases. Besides that, our discussion of the scatter-clustering and heterogeneity is corroborated by the fact that the immune response differs significantly between people, which, again, is consistent with the research of Armitage and Mitchell (2021), who employed cytokine networks to demonstrate that this difference is highly contingent on the host metabolic and genetic factors. The concept in Figures 12 and 13 of late-phase immune suppression contributes to the notions expressed by Feghali and Wright (2020) regarding the decrease of cytokines in chronic inflammatory disease. Finally, the cytokine dysregulation pattern combinations expressed in both anemia and infection in this article demonstrate the multisystemic interaction reported by Sharma et al. (2022). This shows that hematologic abnormalities augment immunological malfunction induced by infection.

Such data indicate that anemia contributes significantly to the cytokine profiles in the course of infection that results in the emergence of a more significant inflammatory state with low immunological control. This not only adds to the worsening of the disease but also complicates the clinical treatment of the disease, this is why the necessity to identify the abnormalities in cytokines in the patients as early as possible and introduce

systematic therapeutic interventions. The study can add to the literature on the subject by having a comprehensive cytokine connotation and predictive model that could help in the risk classification and therapy choices in resource constraining environments. The impact of cytokine surveillance on longterm effects, anti-inflammatory therapy, individually tailored and the impact of micronutrient maintenance on immune homeostasis ought to be included in future research.

CONCLUSION

These findings of this study are a good evidence that the interplay of anemia and infectious diseases results in a specific and exaggerated cytokine-response pattern, with an increase in the number of inflammatory responses, dysfunctional compensatory control signaling, and high-oxidative-stress that leads to a more devastating and dysregulated immune state. The inflammatory cytokine levels are high and the lack of anti-inflammatory modulation evidence high effect of immunological stress because of a combination of two systemic stressors which causes poor clinical outcomes and predisposition to complications. The indicators of the hematologic pattern, pathogen-load indicators, and cytokine profiles demonstrated the manner in which anemia amplified immune activation induced by infections by complicating body acquisition of oxygen, changing the iron utilization process, and worsening oxidative damage. All these are what make the task of the body to react immunologically to respond in a well-coordinated and efficient manner complicated. The anticipatory cytokine-anemia-infection axis formed in this paper supports the notion that such defects of the immune system are not isolated; otherwise, the evidence points to a trend that can be relied upon to forecast the extent of morbidity, levels of risks and transition to the late stage of immune suppression. In addition, the differences between the cluster cytokines and immunological stand out are indicative of the assistance of personalized analysis of patients, since the tasks to different extents are determined by metabolic, genetic and environmental impact. These findings indicate the clinical value of the detection and treatment of anemia in the early stages of treating infectious diseases because the improvement of hematologic stability can directly affect the balance of cytokines, and the total inflammatory load might be minimized. They also describe how cytokine signature surveillance can potentially become a significant aspect of risk classification and treatment decision making in complicated clinical uses. The study provides valuable multisystemic data that anemia is not a comorbidity of infectious diseases exclusively, but an enormous immune modulator, which exacerbates inflammatory cascades and makes clinical prognosis difficult. There is a need to view this interaction as a complex to get the best treatment plans, better patient outcomes, and other researches on cytokine-targeted and hematologic strategies.

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