

IL-6/IL-10 Cytokine Response Patterns and Lymphocyte Subset Profiles During Infectious Episodes in Children With Acute Leukemia: A Retrospective Exploratory Study

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Abstract

Background

Infection during chemotherapy-associated neutropenia is a major clinical problem in children with acute leukemia. Rapid host-response biomarkers may provide complementary information while microbiological results are pending. This study characterized IL-6/IL-10 response patterns and their associations with lymphocyte subsets and microbiological Gram classification using the reported study dataset.

Methods

This single-center retrospective study reviewed 268 clinical episodes recorded between September 2022 and March 2026. A total of 127 infection episodes met the original study criteria. Episodes were described using two neutral cytokine phenotypes: concomitant elevation of IL-6 and IL-10 (dual-elevation profile) and IL-6 elevation with absent or minimal IL-10 elevation (IL-6-predominant profile). Cytokines and lymphocyte subsets were compared between profiles. Concordance with blood culture, sputum culture, and next-generation sequencing findings was examined in microbiologically documented bacterial episodes. Associations with response to meropenem or vancomycin were explored secondarily.

Results

Among 62 neutropenic infection episodes, 32 had a dual-elevation profile and 30 had an IL-6-predominant profile. The dual-elevation group had higher absolute lymphocyte, CD3 + T-cell, CD4 + T-cell, CD8 + T-cell, B-cell, and IL-10 levels (all $P < 0.05$). Similar differences in absolute lymphocyte, CD3+, CD4+, CD8+, B-cell, and IL-10 measures were observed in 65 non-neutropenic episodes. Among 24 microbiologically documented bacterial episodes, 12/14 (85.71%) Gram-negative episodes showed the dual-elevation profile, whereas 9/10 (90.00%) Gram-positive episodes showed the IL-6-predominant profile ($P = 0.001$). In the exploratory treatment-response subset, 27/32 (84.38%) meropenem-responsive episodes showed the dual-elevation profile and 28/31 (90.32%) vancomycin-responsive episodes showed the IL-6-predominant profile ($P < 0.001$).

Conclusions

Distinct IL-6/IL-10 response patterns were associated with different lymphocyte-subset profiles and showed substantial concordance with microbiological Gram classification in this retrospective cohort. Because cytokine information contributed to the original phenotype definition, the findings should be

considered hypothesis-generating and require prospective validation using independently defined microbiological outcomes.

1. Introduction

Contemporary pediatric fever-and-neutropenia guidance treats febrile neutropenia as a medical emergency requiring prompt clinical assessment, blood cultures, risk stratification, and timely empiric antibacterial therapy when indicated [1]. Emergency-care reviews similarly emphasize that children receiving cancer therapy may deteriorate rapidly and that antimicrobial treatment should not be delayed while definitive microbiological results are pending [2]. Recent pediatric oncology reviews continue to identify febrile neutropenia as a major cause of treatment-related morbidity and a setting in which earlier risk discrimination remains clinically important [3]. In a prospective cohort of children with chemotherapy-induced febrile neutropenia, documented infection and bloodstream infection were strongly linked to clinical and laboratory risk features available at presentation [4]. Severe infection also remains an important complication during maintenance treatment of childhood acute lymphoblastic leukemia (ALL), where treatment intensity and host factors can influence infectious risk [5].

Fever is not confined to periods of profound neutropenia. In children receiving therapy for ALL or lymphoblastic lymphoma, infectious events are also common during febrile episodes without severe neutropenia, although bloodstream infection is less frequent than non-bloodstream infectious diagnoses [6]. Management of low-risk chemotherapy-induced febrile neutropenia remains heterogeneous across centers, particularly with respect to outpatient strategies and antibacterial treatment [7]. Prospective multicenter validation of clinical prediction models in febrile non-neutropenic children with cancer further demonstrates that bloodstream-infection risk can be stratified using routinely available clinical variables [8].

Because cultures require time and may remain negative despite clinically important infection, host-response biomarkers have been investigated as adjuncts to routine microbiology. Reviews of immune signatures in pediatric cancer describe cytokines, C-reactive protein (CRP), procalcitonin (PCT), and emerging cellular markers as candidate tools for early risk assessment [9]. A systematic review of immunocompromised children found that CRP, PCT, IL-6, IL-8, IL-10, and TNF-alpha were among the most frequently studied markers associated with bacterial infection, while also highlighting substantial heterogeneity in thresholds and study definitions [10]. A recent review focused specifically on children with chemotherapy-induced severe neutropenia likewise concluded that inflammatory biomarkers may aid early assessment but should be interpreted in clinical context [11]. In children with acute leukemia and febrile neutropenia, IL-6 has shown useful performance for identifying bacteremia [12]. In a large pediatric hematology/oncology cohort, IL-6 and IL-10 were associated with severe infection and septic shock and showed useful predictive performance for selected high-risk outcomes [13]. Conversely, a prospective pediatric fever-and-neutropenia study found that inflammatory biomarkers did not consistently improve discrimination beyond clinical variables, underscoring the need for cautious interpretation [14]. More recently, a pediatric septic-shock study reported that IL-6 and IL-10 differed

according to pathogen classification, with the clearest Gram-related differences observed in bloodstream infection [15].

The cellular immune context may also influence cytokine responses. Longitudinal studies of children treated for acute leukemia have demonstrated substantial and compartment-specific changes in T cells, B cells, and immunoglobulin recovery during and after chemotherapy [16]. A 2024 cohort of children with ALL similarly documented marked treatment-related impairment of T-, B-, and NK-cell compartments, with incomplete recovery of some T-cell measures [17]. Recent multiparameter immunophenotyping further supports persistent abnormalities in CD4 + T-cell, B-cell, NK-cell, and regulatory T-cell compartments across disease and remission states in pediatric ALL [18].

Microbiological methods are also evolving. Metagenomic next-generation sequencing (mNGS) has shown higher pathogen-detection sensitivity than conventional testing in pediatric oncology patients with suspected bloodstream infection and may be particularly useful when routine cultures are unrevealing [19]. Earlier work in pediatric febrile neutropenia likewise demonstrated the feasibility of blood-based next-generation sequencing for pathogen detection, although interpretation requires careful clinical correlation [20]. Against this background, the primary objective of this study was to characterize lymphocyte-subset differences between two IL-6/IL-10 response profiles in neutropenic and non-neutropenic infectious episodes in children with acute leukemia. Secondary objectives were to examine concordance between these profiles and independently obtained microbiological Gram classification and to describe their exploratory association with antibacterial treatment response.

2. Methods

2.1 Study design and setting

This was a single-center retrospective exploratory study of clinical episodes in children with acute leukemia treated at the Department of Pediatrics, The First Affiliated Hospital of Kunming Medical University, China, between September 2022 and March 2026. The source dataset included 268 clinical episodes, of which 127 met the study infection criteria and formed the analytic cohort. The same child could contribute more than one episode when episodes were separated by at least 28 days.

2.2 Participants and clinical definitions

Eligible patients were aged > 1 month and < 18 years and had acute lymphoblastic leukemia or acute myeloid leukemia established by bone-marrow morphology, immunophenotyping, cytogenetics, and molecular testing according to the diagnostic standards used at the study center. Patients with autoimmune disease or primary immune deficiency were excluded, as were episodes attributed to infusion, transfusion, drug-related, or tumor-related fever. Infection was defined in the source protocol by fever (temperature $\geq 37.7^{\circ}\text{C}$) and/or symptoms compatible with infection, such as cough or urinary symptoms. Neutropenia was defined as an absolute neutrophil count $< 0.5 \times 10^9/\text{L}$.

2.3 Cytokine response profiles

The original analysis categorized infection episodes according to the IL-6/IL-10 elevation pattern. To avoid circular diagnostic labeling, neutral phenotype terminology is used in this manuscript: (1) a dual-elevation profile, corresponding to marked elevation of both IL-6 and IL-10; and (2) an IL-6-predominant profile, corresponding to marked IL-6 elevation with absent or only minimal IL-10 elevation. In the microbiological concordance analysis, the reported laboratory reference thresholds were IL-6 > 5.3 pg/mL and IL-10 > 4.91 pg/mL. Episodes were further stratified by the presence or absence of neutropenia.

2.4 Laboratory measurements

At hospital admission, lymphocyte subsets, cytokines, complete blood count, and C-reactive protein were routinely measured. When infection symptoms developed, blood was obtained before antibacterial treatment for lymphocyte subsets, cytokines, procalcitonin, C-reactive protein, complete blood count, blood culture, sputum culture, and/or next-generation sequencing as clinically indicated. The recorded cytokines were IL-2, IL-4, IL-6, IL-10, interferon- γ , and tumor necrosis factor- α . Lymphocyte phenotyping included CD3 + T cells, CD4 + T cells, CD8 + T cells, CD19 + B cells, CD16 + CD56+ natural killer cells, and the CD4+/CD8 + ratio. Fresh venous blood was analyzed using a BD FACSCanto II flow cytometer (BD Biosciences, USA).

2.5 Microbiological concordance and exploratory treatment-response analyses

Blood culture, sputum culture, and next-generation sequencing results were used as microbiological evidence. The source analysis reported 24 bacterial episodes with a documented Gram classification: 14 Gram-negative and 10 Gram-positive. Concordance was calculated between microbiological Gram classification and the two cytokine response profiles. In a separate exploratory analysis, episodes in which body temperature normalized within 72 hours after treatment were classified as responsive to meropenem or vancomycin. Cytokine response patterns were compared between 32 meropenem-responsive and 31 vancomycin-responsive episodes. Because antimicrobial response is influenced by infection site, organism susceptibility, co-therapy, and host factors, this analysis was treated as exploratory rather than as a microbiological reference standard.

2.6 Statistical analysis

Continuous variables with an approximately normal distribution were summarized as mean $\pm$ standard deviation and compared using independent-samples t tests. Non-normally distributed variables were summarized as median (interquartile range) and compared using rank-sum tests. Categorical variables were summarized as n (%) and compared using χ^2 tests. Spearman correlation was used for non-normally distributed variables when applicable in the original analysis. All P values were two-sided, and $P < 0.05$ was considered statistically significant. The present manuscript reports the statistical results available from the original study analysis and does not introduce additional modeling or recalculated diagnostic-performance estimates.

3. Results

3.1 Study cohort

The source dataset contained 268 clinical episodes. A total of 127 infectious episodes met the original analytic criteria. Among these, 62 neutropenic episodes were categorized as 32 dual-elevation and 30 IL-6-predominant episodes, while 65 non-neutropenic episodes were categorized as 35 dual-elevation and 30 IL-6-predominant episodes. The study flow is summarized in Fig. 1.

3.2 Cytokine and lymphocyte-subset profiles during neutropenic infection

Among neutropenic infection episodes, absolute lymphocyte counts, absolute CD3 + T-cell counts, absolute CD4 + T-cell counts, absolute CD8 + T-cell counts, absolute B-cell counts, and IL-10 concentrations were higher in the dual-elevation profile than in the IL-6-predominant profile (all $P < 0.05$). IL-4, IL-6, IFN- γ , TNF- α , the corresponding lymphocyte-subset percentages, NK-cell measures, and the CD4+/CD8 + ratio did not differ significantly (Table 1).

Table 1
Cytokine and lymphocyte-subset characteristics of neutropenic infection episodes

Variable	Dual-elevation profile (n = 32)	IL-6-predominant profile (n = 30)	Z	P
IL-4 (pg/mL)	0.45 (0.10–0.93)	0.62 (0.36–0.98)	–0.927	0.354
IL-6 (pg/mL)	24.945 (7.04–71.99)	105.3 (29.18–348.67)	–1.941	0.052
IL-10 (pg/mL)	20.14 (8.85–81.90)	3.27 (2.58–9.15)	–3.300	0.001
IFN- γ (pg/mL)	7.84 (1.43–23.64)	3.36 (2.06–17.48)	–0.340	0.734
TNF- α (pg/mL)	1.12 (0.70–1.78)	0.96 (0.47–1.04)	–1.529	0.126
Absolute lymphocytes (cells/ μ L)	526 (356–1321.75)	190 (36–461.5)	–3.106	0.002
CD3 + T cells (%)	85.3 (74.01–92.74)	91.09 (80.7–96.20)	–1.019	0.308
Absolute CD3 + T cells (cells/ μ L)	434 (288–1063.75)	183 (25–435.5)	–3.009	0.003
CD8 + T cells (%)	34.41 (23.65–55.83)	28.41 (21.42–49.02)	–0.388	0.698
Absolute CD8 + T cells (cells/ μ L)	221 (139.75–369)	86 (7–204)	–2.815	0.005
CD4 + T cells (%)	38.08 (27.92–54.99)	42.76 (30.1–47.97)	–0.146	0.884
Absolute CD4 + T cells (cells/ μ L)	149 (120–663.75)	80 (11.5–208.5)	–2.766	0.006
NK cells (%)	7.885 (3.96–12.72)	4.7 (3.32–12.94)	–0.534	0.593
Absolute NK cells (cells/ μ L)	62 (21.25–110.5)	50 (42–60)	–0.598	0.699
B cells (%)	1.34 (0.07–11.93)	0.21 (0.02–2.33)	–1.677	0.094
Absolute B cells (cells/ μ L)	7 (0–159.25)	1 (0–4)	–2.213	0.027
CD4+/CD8 + ratio	1.26 (0.58–2.05)	1.04 (0.60–2.51)	–0.024	0.981

Note. Values are median (interquartile range) unless otherwise indicated. Absolute lymphocyte-subset counts are reported as cells/ μ L. Cytokines are reported as pg/mL.

3.3 Cytokine and lymphocyte-subset profiles during non-neutropenic infection

Among non-neutropenic infection episodes, the dual-elevation profile was associated with higher absolute lymphocyte counts, absolute CD3 + T-cell counts, absolute CD4 + T-cell counts, absolute CD8 + T-cell counts, absolute B-cell counts, B-cell percentage, and IL-10 concentrations. The CD3 + T-cell percentage was lower in the dual-elevation profile. IL-4, IL-6, IFN- γ , TNF- α , CD8 + percentage, NK-cell measures, and the CD4+/CD8 + ratio did not differ significantly (Table 2).

Table 2
Cytokine and lymphocyte-subset characteristics of non-neutropenic infection episodes

Variable	Dual-elevation profile (n = 35)	IL-6-predominant profile (n = 30)	Z	P
IL-4 (pg/mL)	0.52 (0.27–0.88)	0.50 (0.36–0.98)	–0.165	0.869
IL-6 (pg/mL)	17.77 (9.37–106.54)	53.61 (17.27–121.64)	–0.989	0.323
IL-10 (pg/mL)	18.49 (8.44–35.93)	3.50 (1.09–6.84)	–3.297	0.001
IFN-γ (pg/mL)	6.57 (1.65–28.62)	5.28 (1.32–107.63)	–0.198	0.843
TNF-α (pg/mL)	1.08 (0.65–1.39)	0.73 (0.46–1.38)	–0.660	0.510
Absolute lymphocytes (cells/μL)	1426 (1111–4596)	510 (210–941.5)	–2.703	0.007
CD3 + T cells (%)	76.39 (55.14–89.63)	93.20 (86.89–98.40)	–2.638	0.008
Absolute CD3 + T cells (cells/μL)	1074 (784.5–2300.75)	506 (191.5–885.25)	–2.605	0.009
CD8 + T cells (%)	30.00 (23.47–46.76)	36.00 (17.53–50.44)	–0.396	0.692
Absolute CD8 + T cells (cells/μL)	592 (346.25–1135)	178 (90.5–378)	–2.638	0.008
CD4 + T cells (%)	33.64 (26.15–42.16)	42.38 (35.74–60.77)	–2.242	0.025
Absolute CD4 + T cells (cells/μL)	579 (327.75–902.5)	201 (82–520.75)	–2.144	0.032
NK cells (%)	7.66 (3.37–15.41)	6.15 (0.71–11.12)	–0.989	0.323
Absolute NK cells (cells/μL)	154 (69.75–708)	117 (112.5–153)	–0.737	0.396
B cells (%)	10.74 (0.47–19.80)	0.18 (0.07–0.40)	–2.144	0.032
Absolute B cells (cells/μL)	133 (13.25–1435)	1 (0–4.25)	–3.198	0.001
CD4+/CD8 + ratio	0.91 (0.6925–1.3975)	1.20 (0.6825–3.545)	–0.759	0.448
Note. Values are median (interquartile range). Absolute lymphocyte-subset counts are reported as cells/μL. Cytokines are reported as pg/mL.				

3.4 Concordance with microbiological Gram classification

Among 24 bacterial episodes with reported microbiological Gram classification, 12 of 14 Gram-negative episodes (85.71%) showed the dual-elevation profile and 2 (14.29%) showed the IL-6-predominant profile. Among 10 Gram-positive episodes, 1 (10.00%) showed the dual-elevation profile and 9 (90.00%) showed the IL-6-predominant profile (P = 0.001; Table 3). The documented microbiological results comprised 11 blood-culture-positive episodes (7 Gram-negative, 4 Gram-positive), 7 sputum-culture-positive episodes (4 Gram-negative, 3 Gram-positive), and 6 next-generation-sequencing-positive episodes (3 Gram-negative, 3 Gram-positive).

Table 3
Concordance between microbiological Gram classification and IL-6/IL-10 response profile

Microbiological classification	Dual-elevation profile	IL-6-predominant profile	P
Gram-negative bacteria (n = 14)	12 (85.71%)	2 (14.29%)	0.001
Gram-positive bacteria (n = 10)	1 (10.00%)	9 (90.00%)	

3.5 Exploratory antibacterial treatment-response analysis

Among episodes classified as clinically responsive within 72 hours, 27 of 32 (84.38%) meropenem-responsive episodes showed the dual-elevation profile, whereas 28 of 31 (90.32%) vancomycin-responsive episodes showed the IL-6-predominant profile ($\chi^2=35.223$, P < 0.001; Table 4). These associations are descriptive and do not establish that the cytokine profile should be used to select a specific antibiotic.

Table 4
Exploratory association between IL-6/IL-10 response profile and antibacterial treatment-response group

Treatment-response group	Dual-elevation profile	IL-6-predominant profile	χ^2	P
Meropenem-responsive (n = 32)	27 (84.38%)	5 (15.62%)	35.223	< 0.001
Vancomycin-responsive (n = 31)	3 (9.68%)	28 (90.32%)		

Note. Response was defined in the source study as normalization of body temperature within 72 hours. This analysis is hypothesis-generating and should not be used to infer pathogen identity or comparative drug efficacy.

4. Discussion

In this retrospective exploratory study, IL-6/IL-10 response phenotypes were accompanied by distinct lymphocyte-subset patterns in children with acute leukemia and infection. In both neutropenic and non-neutropenic episodes, the dual-elevation profile was generally associated with higher absolute lymphocyte and major T-cell subset counts than the IL-6-predominant profile. In the limited subset with

reported microbiological Gram classification, the dual-elevation profile was common in Gram-negative episodes, whereas the IL-6-predominant profile was common in Gram-positive episodes. These findings support an association between host cytokine-response patterns, cellular immune profiles, and pathogen characteristics rather than a fully validated diagnostic test.

The microbiological concordance is biologically plausible and aligns with several independent studies. In patients with lymphoma during myelosuppression, Zhu et al. reported higher IL-6 and IL-10 concentrations in Gram-negative infection and described distinct IL-6/IL-10 cytokine patterns for Gram classification [21]. In a microbiologically documented pediatric sepsis cohort, Zhang et al. also found significantly higher IL-6 and IL-10 concentrations in Gram-negative than Gram-positive sepsis [22]. In a prospective pediatric cancer study, Xu et al. found that IL-6 and IL-10 were associated with high-risk febrile illness and performed better than some conventional inflammatory markers for selected outcomes [23]. Earlier work in pediatric hematology/oncology patients with septic shock similarly identified higher IL-6, IL-10, and TNF-alpha concentrations in Gram-negative bacteremia and highlighted IL-10 as a useful discriminator [24]. Tang et al. also described distinct Th1/Th2 cytokine profiles between Gram-negative and Gram-positive bacteremia in pediatric hematology/oncology patients [25]. In another pediatric cancer febrile-neutropenia cohort, elevated IL-10 and PCT at presentation were associated with bacteremia, supporting the potential value of combining cytokine and conventional biomarker information [26].

These similarities should not be interpreted as proof that one cytokine threshold can reliably identify bacterial class across settings. PCT concentrations can vary with pathogen type and infection focus, illustrating the broader context dependence of inflammatory biomarkers [27]. Pathogen distributions in pediatric febrile neutropenia also vary by institution and over time, which is why empiric antibacterial choices should remain aligned with local epidemiology and susceptibility patterns [28]. Interpretation of mNGS is likewise context dependent; recent pediatric hematology/oncology data indicate that neutrophil count can influence the clinical meaning of detected organisms [29]. Clinical variables therefore remain important, and earlier pediatric febrile-neutropenia work has shown that severe-infection risk is related to conventional features such as fever intensity, CRP, and underlying disease rather than to a single laboratory marker alone [30]. The present findings are best viewed as complementary host-response information rather than a replacement for microbiological testing or clinical judgment.

The lymphocyte-subset findings should also be interpreted as host-response characteristics rather than pathogen-specific mechanisms. Acute leukemia and chemotherapy can alter CD4+, CD8+, B-cell, and NK-cell compartments substantially, and those effects vary with treatment phase and immune recovery. The lower absolute CD3+, CD4+, CD8+, and B-cell counts observed in the IL-6-predominant neutropenic phenotype may therefore reflect a different state of treatment-related immune suppression or marrow recovery. Because chemotherapy phase, corticosteroid exposure, leukemia subtype, and timing relative to marrow recovery were not modeled in the source analysis, causal or mechanistic conclusions cannot be drawn.

The exploratory association with meropenem- and vancomycin-responsive episodes is noteworthy but should not be interpreted as evidence for biomarker-directed antibiotic selection. Fever resolution within 72 hours is not a pathogen-specific reference standard, and treatment response can be affected by co-administered antimicrobials, infection source, susceptibility, source control, and natural clinical course. At most, this result generates a hypothesis that cytokine response phenotypes might eventually contribute to early antimicrobial stratification when integrated with local epidemiology, patient risk, and microbiological data.

This study has several limitations. First, it was retrospective and single-center. Second, cytokine information contributed to the original phenotype definition; consequently, the study is not a formal diagnostic-accuracy study of IL-6 or IL-10. Third, only 24 episodes had a reported microbiological Gram classification, and these included blood culture, sputum culture, and next-generation sequencing rather than a uniform reference standard. Fourth, the same child could contribute more than one episode, so observations may not be statistically independent. Finally, no external validation cohort was available. Prospective validation in independently microbiologically defined infection cohorts is therefore required.

5. Conclusions

In children with acute leukemia and infectious episodes, IL-6/IL-10 cytokine response patterns were associated with distinct lymphocyte-subset profiles. In the limited microbiologically documented subset, concomitant IL-6/IL-10 elevation was more common in Gram-negative infection, whereas an IL-6-predominant pattern was more common in Gram-positive infection. These findings are hypothesis-generating and support prospective studies that define Gram status independently of cytokine measurements and evaluate whether integrated cytokine and lymphocyte-subset profiling can improve early clinical risk stratification.

Abbreviations

ALL
acute lymphoblastic leukemia
ANC
absolute neutrophil count
CRP
C-reactive protein
IFN- γ
interferon gamma
IL-2
interleukin-2
IL-4
interleukin-4
IL-6

interleukin-6
IL-10
interleukin-10
mNGS
metagenomic next-generation sequencing
NK
natural killer
PCT
procalcitonin
TNF- α
tumor necrosis factor alpha
CD
cluster of differentiation.

Declarations

Ethics approval and consent to participate

Ethical approval for this study was obtained from the Ethics Committee of the First Affiliated Hospital of Kunming Medical University (Ethics Approval No. L-2026-183). The study was conducted in accordance with the Declaration of Helsinki and relevant national ethical guidelines. All data were anonymized before analysis, and no information capable of identifying individual participants was retained. The requirement for informed consent to participate was waived by the Ethics Committee of the First Affiliated Hospital of Kunming Medical University because of the retrospective nature of the study and the use of anonymized clinical data.

Consent for publication

Not applicable. The manuscript does not contain any identifiable individual participant data.

Competing interests

The authors declare that they have no competing financial or non-financial interests.

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Author Contribution

HD and LM designed the study. YZ and XS made revisions to the manuscript. YZ provided the foundation and support for this study. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

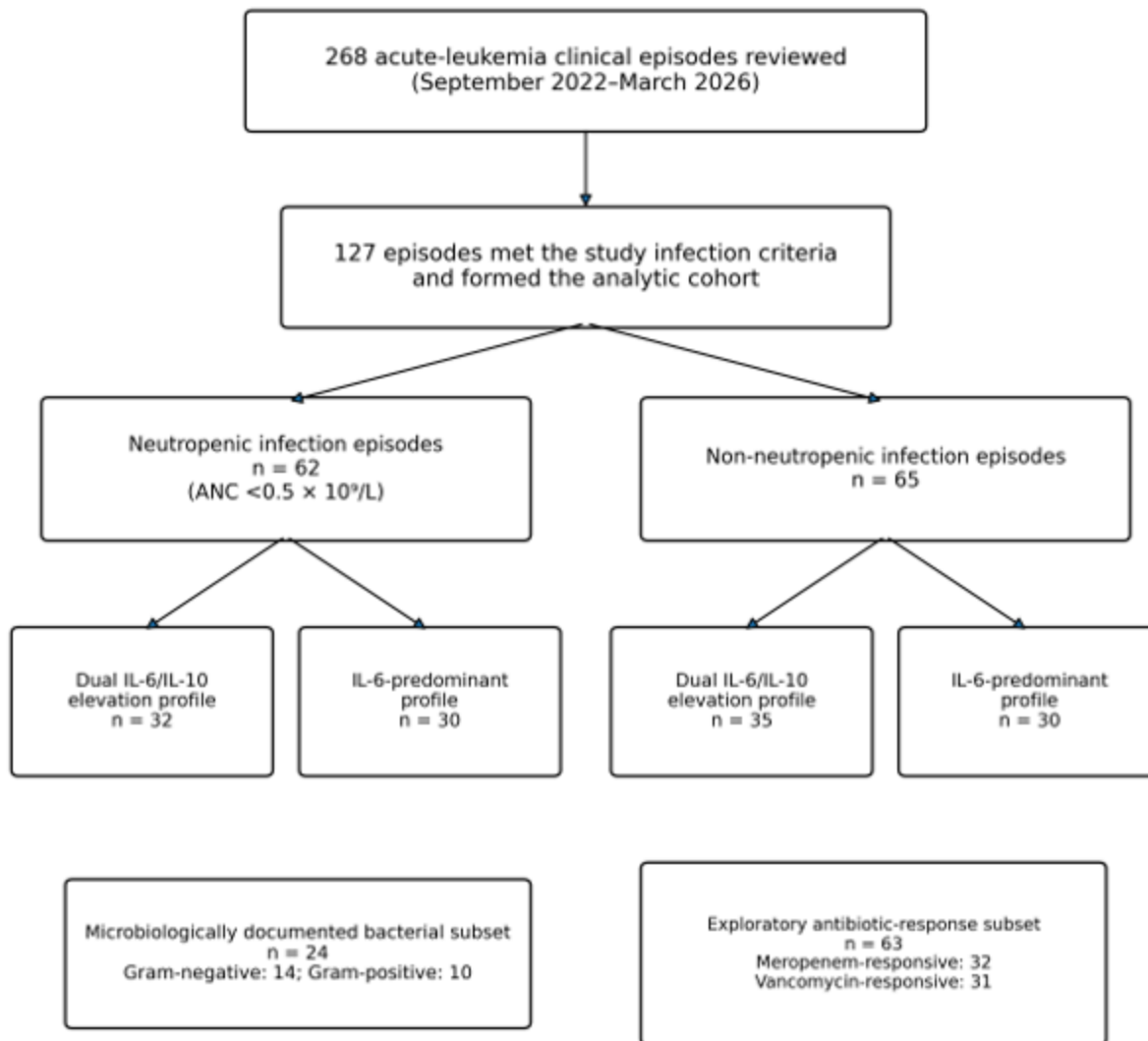
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Figures



Subsets overlap with the 127 infection episodes; they are shown as secondary analyses.

Figure 1

Study flow based on the episode counts reported in the source study. The same child could contribute more than one episode if episodes were separated by at least 28 days.

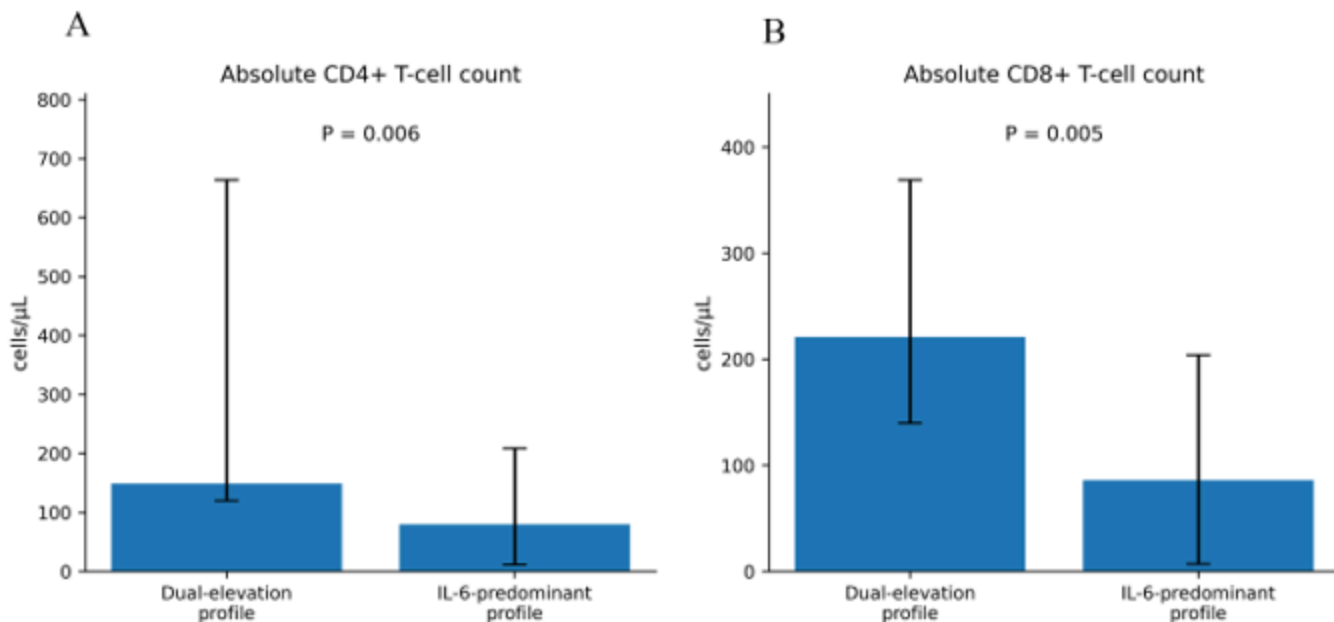


Figure 2

(A) Absolute CD4+ T-cell counts and (B) absolute CD8+ T-cell counts in neutropenic infection episodes according to cytokine response profile. Bars represent the reported medians, and error bars represent the reported interquartile ranges. Individual-level data were not available for replotting.

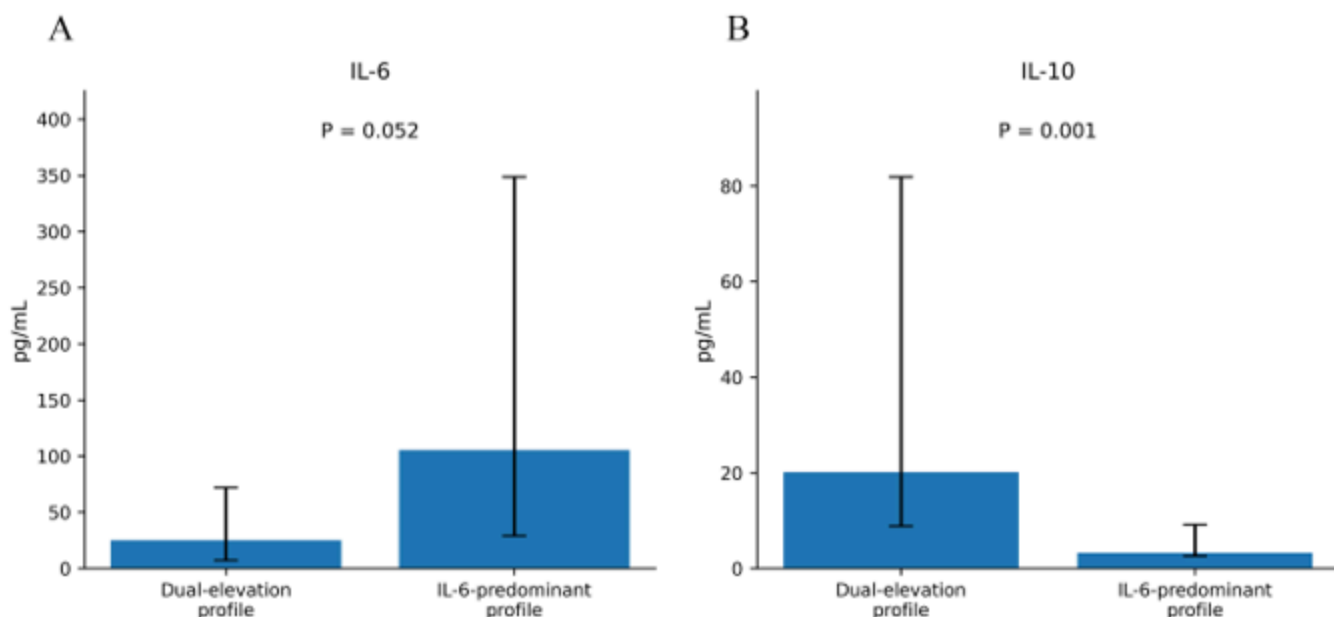


Figure 3

(A) IL-6 concentrations and (B) IL-10 concentrations in neutropenic infection episodes according to cytokine response profile. Bars represent the reported medians, and error bars represent the reported

interquartile ranges. Individual-level data were not available for replotting.

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