Cytopenias and infections following ciltacabtagene autoleucel in heavily pretreated relapsed or refractory multiple myeloma

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Abstract

Ciltacabtagene autoleucel (cilta-cel) was approved by the Food and Drug Administration in February 2022 for the treatment of relapsed/refractory multiple myeloma after four lines of therapy. On the CARTITUDE-1 trial, grade ≥3 cytopenias and infections were common. Herein, we sought to characterize cytopenias and infections after cilta-cel infusion in the standard-of-care setting. This multicenter, retrospective study included 105 patients who received cilta-cel; 91 reached day 90 and 49 reached day 180 of follow-up. Grade ≥3 cytopenia was present among 52% of patients on day 30, and 24% of patients on day 90. Based on the newer immune effector cell-associated hematotoxicity (ICAHT) grading for neutropenia severity, 11 patients (10%) experienced grade ≥3 early ICAHT in the first 30 days, while only three (3.3%) experienced grade ≥3 late ICAHT after day 30. On univariate analysis, any grade thrombocytopenia at apheresis was associated with grade ≥3 cytopenia at both days 30 and 90. Granulocyte colony-stimulating factor was administered to 65%, transfusion support to 38%, thrombopoietin agonists to 10%, intravenous immunoglobulins to 52%, and $CD34^+$ stem cell boosts to 9.5% of patients. Infections occurred in 49% of patients and were severe in 32%. Earlier infections in the first 30 days were equally bacterial (42%) and viral (42%). Later infections between days 31-100 and after day 100 were mostly viral (59% and 60%, respectively), with only 32% and 12% being grade ≥3 in each time period. On univariate analysis, worse Eastern Cooperative Oncology Group performance status at lymphodepletion, higher maximum grade of cytokine-release syndrome, delayed neurotoxicity, steroid and anakinra use, and lower IgA levels at day 90 were associated with severe infections.

Introduction

Multiple myeloma is an incurable plasma cell neoplasm despite recent advancements in therapeutic modalities. The proclivity for relapse and refractoriness to conventional plasma cell-directed treatments, including proteasome inhibitors, immunomodulatory agents, and anti-CD38 antibodies, underscores the challenging clinical landscape marked by inferior outcomes.[1-4] Ciltacabtagene autoleucel (cilta-cel) is a second-generation autologous B-cell maturation antigen (BCMA)-targeting chimeric antigen receptor (CAR) T-cell therapy that incorporates dual camelid sites, providing high avidity against the BCMA antigen.[5-8] In February 2022, cilta-cel became the second CAR T-cell therapy to receive approval from the US Food and Drug Administration (FDA) for relapsed or refractory multiple myeloma (RRMM) after exposure to four or more prior lines of therapy.[9] This approval was based on the CARTITUDE-1 trial, which demonstrated a 98% overall response rate, 83% stringent complete responses, and a median progression-free sur-

vival of 34.9 months.[5,10,11]

Despite these remarkable efficacy outcomes, cilta-cel has been linked to some serious and potentially life-threatening toxicities. Cytopenias were noted to be the most common treatment-emergent adverse event in the phase Ib/II CARTITUDE-1 trial, including grade ≥3 neutropenia (95%), grade ≥3 anemia (68%), and grade ≥3 thrombocytopenia (60%), with approximately 90% and 60% of grade ≥3 neutropenia and thrombocytopenia, respectively, resolving to grade ≤2 by day 60.[5,11] These adverse events were partially attributed to lymphodepleting chemotherapy with fludarabine and cyclophosphamide given before the cilta-cel infusion. Cytopenias were managed with growth factors and blood product transfusion as per institutional guidelines.[5,9] Infections were also noted in the majority of patients (58%); these were grade ≥3 in 20%, and resulted in death in 4% of patients. Upper respiratory tract infections were most common overall (16%), with the most frequent grade ≥3 infections being pneumonia (8%) and sepsis (4%).[5]

Although infections and cytopenias are well-known sequelae related to myelosuppression after cilta-cel administration, longitudinal data on duration and recovery of cytopenias are limited. In this retrospective multicenter study, we evaluated cellular and humoral reconstitution, infections, and the requirement for supportive interventions in response to treatment-related cytopenias among patients with RRMM treated with standard-of-care cilta-cel, prior to its FDA approval in the second-line setting. The goal was to enhance our understanding of the short- and long-term safety of cilta-cel, particularly for patients who would have been excluded from clinical trials due to comorbidities or preexisting cytopenias, which may help to optimize supportive care strategies and improve patients' overall outcomes.

Methods

Patients and data collection

This was a multi-institutional retrospective analysis of consecutive adult patients with RRMM who underwent treatment with standard-of-care cilta-cel after four or more prior lines of therapy between May 11, 2022, and May 30, 2023. Each participating center obtained independent institutional review board approval, which included a waiver of informed consent. The study adhered to the principles of the Declaration of Helsinki, and the data abstraction was performed by trained personnel. Data extracted from patients' records included demographics, baseline disease status and prior therapies, complete blood counts, multiple myeloma laboratory tests, bone marrow biopsy reports, infection data, and supportive medication administration. Patients underwent apheresis following institutional procedures between March 11, 2022, and March 13, 2023. Cytopenia data were collected at the following timepoints: at apheresis, before lymphodepletion, day 0, day 7, day 14, day 21, day 30, day 90, and day 180 after infusion.

Definitions and clinical assessment

Cytopenias were graded according to Common Terminology Criteria for Adverse Events (CTCAE) version 5.0[12] and immune effector cell-associated hematotoxicity (ICAHT).[13] Immune-mediated toxicities were graded by the American Society of Transplantation and Cellular Therapy (ASTCT) consensus criteria.[14] Response was assessed based on the International Myeloma Working Group criteria.[15] Antimicrobial prophylaxis was provided to all patients according to institutional guidelines as previously published.[16]

Neutropenia was defined as per CTCAE v5.0, with grade 1 signifying an absolute neutrophil count (ANC) less than the lower limit of normal, or <1,800 cells/μL based on the clinical laboratory. Neutropenia was designated as grade ≥3 if the ANC was less than 1,000 cells/μL, as per CTCAE criteria. Neutrophil recovery was defined as the first day of ANC greater than 500 cells/μL sustained on two consecutive days without granulocyte colony-stimulating factor (G-CSF) use in the preceding 7 days.

An infection was defined as any instance of viral, bacterial, or fungal disease identified by microbiological data, radiographic findings, or clinical symptoms in the retrospective chart review, censored at day 100. Severe infection was defined as an infection requiring intravenous antibiotics and/or hospitalization, which by CTCAE criteria would be grade 3 or higher. Infections diagnosed outside of the institution were classified based on patients' reports and available medical records. The onset of fever following CAR T-cell infusion without microscopic, radiographic or symptomatic evidence of infection was attributed to cytokine release syndrome (CRS) and not counted as an infection. Although there were some differences based on institutional protocols, overall infectious prophylaxis strategies were similar across all centers in our study, and in accordance with previously published guidelines (*Online Supplementary Tables SA-SE*).[17,18]

All definitions of immunity were based on clinical laboratory definitions. For herpes simplex virus (HSV) 1/2 immunoglobulin G (IgG), an antibody titer ≤0.89 was considered negative and titer ≥1.10 was considered positive (immune); a titer ≥0.90 and ≤1.09 was indeterminate. For varicella-zoster virus (VZV) IgG antibodies, results were reported as positive (immune), negative, or equivocal. Cytomegalovirus (CMV) IgG antibodies were reported as positive (immune) or negative. Pneumococcal testing comprised 14 separate IgG antibody titers, and an antibody concentration >1.0 μg/mL was considered long-term protection (immunity).

Statistical analysis

Associations between patients' characteristics and grade ≥3 cytopenias at days 30 and 90 were evaluated using Wilcoxon rank sum for continuous variables as well as Pearson χ^2 and Fisher exact tests for categorical variables. Box and

whisker plots were used to illustrate trends in inflammatory markers from apheresis to day 180. Time-series plots were used to visualize cellular and humoral immune reconstitution from apheresis to day 180. Kruskal-Wallis rank sum tests were applied to assess differences in inflammatory markers and cellular and humoral reconstitution over time. Time-to-event analysis was used to model infection density. Evolution of viral titers and pneumococcal titers from apheresis to day 180 were illustrated using alluvial plots[19,20] and a heatmap,[21] respectively. Statistical tests were two-sided and a $P<0.05$ was considered statistically significant. All statistical analyses were conducted using R version 4.1.2. and GraphPad Prism version 8.0.2.

Although underpowered, we performed an exploratory multivariable logistic regression analysis of risk factors associated with grade ≥3 cytopenia at day 90. The *a priori* risk factors assessed included the number of prior lines of therapy (continuous, per 1 line of therapy), high marrow burden (≥50%; yes, no), and grade ≥3 cytopenia at day -5 (yes, no). A forest plot was used to depict each odds ratio and 95% confidence interval from the multivariable analysis.

Results

Patients' characteristics

The study included a total of 105 patients, all of whom underwent apheresis followed by lymphodepleting chemotherapy and cilta-cel infusion (Figure 1). The patients' baseline characteristics are described in Table 1. The median time from apheresis to cilta-cel infusion was 69.5 days (*Online Supplementary Table S1*). All patients had received at least four prior lines of therapy. Among them, 77 (73%) were triple-class refractory, and 88 (84%) had a history of autologous stem cell transplant. When gain of 1q was included as a high-risk cytogenetic feature, 64 (72%) patients were classified as having high-risk cytogenetics. Lymphodepleting chemotherapy consisted of fludarabine and cyclophosphamide, as in CARTITUDE-1,[8,9] in 82% of the cohort of patients, cladribine/cytarabine in 7%, single-agent cyclophosphamide in 6%, and bendamustine in 6%. Most patients included in this cohort (45%) would not have been considered eligible for the pivotal CARTITUDE-1 trial. Thirty-two (36%) patients had high CAR-HEMATOTOX scores at baseline before cilta-cel infusion.[22,23] The median follow-up from cilta-cel infusion was 6 months.

Safety and response outcomes

The overall response rate was 92%, with 64 (63%) patients achieving complete response or better as the best response in the first 90 days after cilta-cel infusion (*Online Supplementary Table S2*). Immune-mediated toxicity from treatment was manageable, with grade ≥3 CRS and grade ≥3 immune effector cell-associated neurotoxicity syndrome each occurring in three patients (3%). Analysis of inflammatory markers showed that the level of C-reactive protein was highest at day 7 after infusion, while ferritin and lactate dehydrogenase levels peaked at day 14 after infusion (*Online Supplementary Figure S1*). Fourteen patients (13%) experienced delayed neurotoxicity, including parkinsonian-like syndrome (2.9%), Bell's palsy (5.7%), polyneuropathy (2.9%), and posterior reversible encephalopathy syndrome (1.9%).

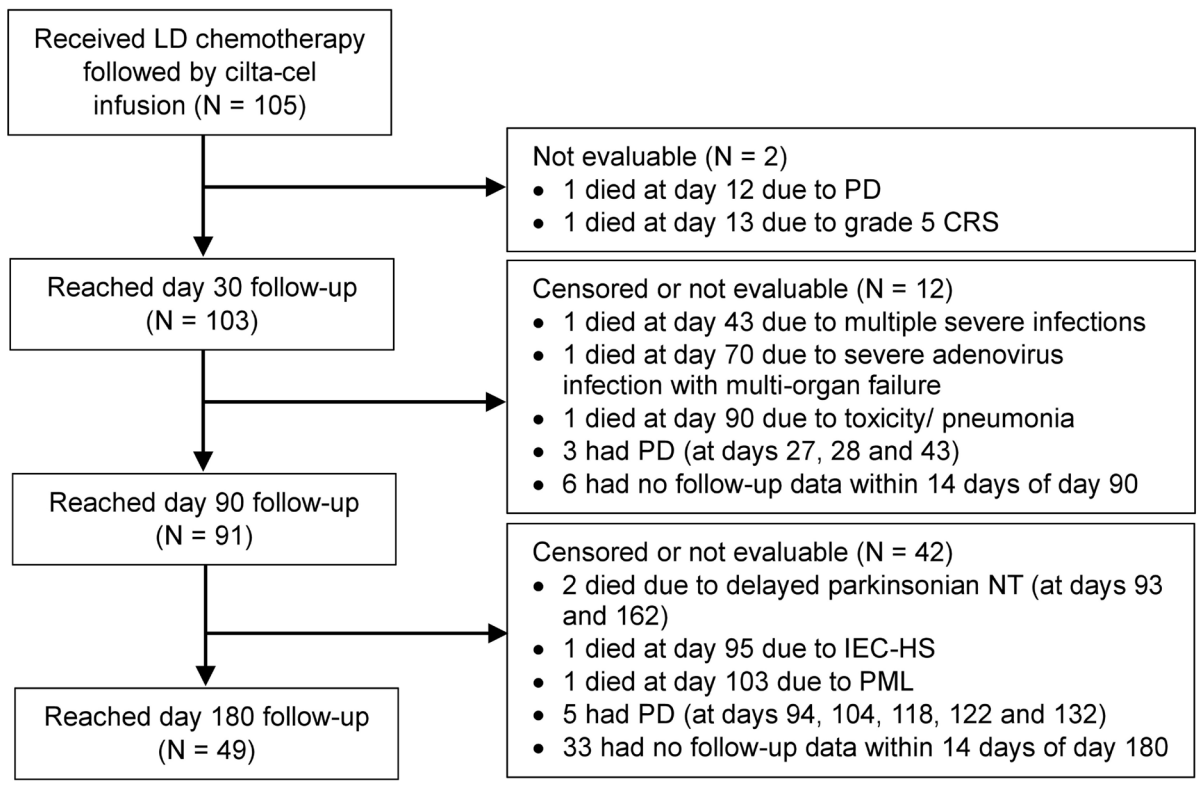

Figure 1. Patient flow diagram. Patients were censored at the time of death or 7 days after progressive disease. LD: lymphodepleting; cilta-cel: ciltacabtagene autoleucel; PD: progressive disease; CRS: cytokine-release syndrome; NT: neurotoxicity; IEC-HS: immune effector cell-associated hemophagocytic lymphohistiocytosis-like syndrome; PML: progressive multifocal leukoencephalopathy.

A total of 59 patients (56%) received tocilizumab and 42 (40%) received steroids for adverse event management. Immune effector cell-associated hemophagocytic lymphohistiocytosis-like syndrome[24] was diagnosed clinically in two patients (2%).

Cytopenias

Though cytopenias were common at baseline with >80% of patients having any-grade cytopenia at both apheresis and day -5, grade ≥3 cytopenias were more commonly seen after lymphodepletion. In detail, cytopenias were most prevalent at day 7 after cilta-cel, with 104 patients (99%) experiencing any-grade cytopenia and 72 (69%) experiencing at least one grade ≥3 cytopenia, based on CTCAE criteria (Table 2A). Notably, on day 7 after cilta-cel, severe neutropenia and anemia had reached their peak prevalence (59% and 21%, respectively). Grade ≥3 cytopenia of any kind persisted in 54 patients (52%) at day 30, and the median platelet count was lowest at day 30 (Figure 2). While cytopenias generally improved over time, there were 12 patients (13%) at day 90 and seven (14%) at day 180 with prolonged, severe (grade ≥3) neutropenia. The prevalence of grade ≥3 cytopenias and recovery over time is shown graphically in Figure 3. Based on the newer ICAHT grading for neutropenia severity, 11 patients (10%) experienced grade ≥3 early ICAHT in the first 30 days after cilta-cel infusion, while only three (3.3%) experienced grade ≥3 late ICAHT after day 30 (Table 2B).

We also examined bone marrow biopsy findings before and after cilta-cel (Table 3). While the proportion of patients with marrow hypocellularity did increase from baseline (16%) to day 30 (33%), the majority of patients had normocellular marrow findings at each timepoint, ranging from 39% of patients at day 30 to 50% at day 180. The median marrow cellularity remained 30-40% throughout the time studied. The median CD138$^+$ plasma cell percentage in the bone marrow decreased to 0% at both timepoints. There were no significant findings to suggest marrow fibrosis or dysplasia.

We conducted univariable analyses to assess risk factors for any cytopenia of grade ≥3 (by CTCAE criteria). Patients with the following characteristics were more likely to have grade ≥3 cytopenia (any lineage) at day 30: baseline extramedullary disease (P=0.016), high-risk cytogenetics (not including gain 1q, P=0.028; including gain 1q, P=0.045), receipt of more than four prior lines of therapy (P=0.026), any-grade anemia or thrombocytopenia at apheresis (P<0.001 and P=0.001, respectively), any-grade cytopenia before lymphodepletion (P=0.021), high CAR-HEMATOTOX score (P=0.006) and tocilizumab use (P=0.001) (*Online Supplementary Tables S3* and *S4*). Patients who developed early ICAHT were more likely to have baseline cytopenias (at apheresis [P=0.002] or prior to lymphodepletion [P=0.047]), earlier onset of CRS (P<0.006), earlier maximum CRS grade (P=0.004), longer CRS duration (P=0.003), and tocilizumab use (P=0.005) (*Online Supplementary Tables S5* and *S6*).

We also conducted a similar analysis for patients who had ongoing grade ≥3 cytopenia at day 90 after cilta-cel. Patients

Table 1. Patients' baseline characteristics prior to ciltacabtagene autoleucel.

Characteristic	N=105
Follow-up time from infusion, months, median (range)	6.0 (0.4-13.5)
Age, years, median (range) ≥70 years, N (%)	63.0 (30.0-76.0) 23 (22)
Male sex, N (%)	58 (55)
Extramedullary disease, N (%) Unknown	31 (30) 1
High marrow burden (≥50%), N (%) Unknown	18 (18) 3
ECOG performance status at lymphodepletion, N (%) 0-1 ≥ 2 Unknown	 97 (94) 6 (5.8) 2
R-ISS at CAR T-cell infusion, N (%) I II III Unknown	 21 (23) 44 (48) 26 (29) 14
High-risk cytogenetics (not including gain 1q), N (%)	43 (49)
High-risk cytogenetics (including gain 1q), N (%) Deletion 17p at infusion Unknown t(4:14) at infusion Unknown t(14:16) at infusion Unknown Gain/amp 1q21 Unknown	64 (72) 27 (29) 11 14 (15) 14 6 (7.6) 26 48 (53) 15
Bridging therapy, N (%)	91 (87)
Prior autologous SCT, N (%)	88 (84)
Prior allogeneic SCT, N (%)	0
Prior BCMA-directed therapy, N (%)	9 (8.6)
Refractoriness to prior treatments, N (%) Refractory to immunomodulatory agent Refractory to proteasome inhibitor Refractory to anti-CD38 monoclonal antibody Double-refractory[a] Triple-refractory[b] Penta-refractory[c]	 91 (87) 91 (87) 96 (91) 82 (78) 77 (73) 28 (27)
Eligible for CARTITUDE trial, N (%)	47 (45)
CAR-HEMATOTOX score, N (%) Low High Unknown	 57 (64) 32 (36) 16

[a]Double-refractory disease: refractory to an immunomodulatory agent (IMiD) and a proteasome inhibitor (PI). [b]Triple-refractory disease: refractory to an IMiD, PI and daratumumab. [c]Penta-refractory disease: refractory to lenalidomide, pomalidomide, bortezomib, carfilzomib and daratumumab. ECOG: Eastern Cooperative Oncology Group; R-ISS: Revised International Staging System. CAR: chimeric antigen receptor; SCT: stem cell transplantation; BCMA: B-cell maturation antigen.

with grade ≥3 cytopenia at this point were more likely to have been more heavily pre-treated with triple-class refractory disease (*P*=0.03) and have had any-grade thrombocytopenia at apheresis (*P*=0.022) (*Online Supplementary Table S7*). When evaluating cytopenias using the ICAHT definition, those with late ICAHT (defined as neutropenia observed beyond day 30 with the severity [grade 1-4] defined by the depth of neutropenia) were more likely to have had baseline high-risk cytogenetics, triple-class refractory disease and more likely to have been ineligible for the pivotal CARTITUDE-1 trial (*Online Supplementary Table S8*). We conducted an exploratory multivariable logistic regression analysis of selected risk factors associated with grade ≥3 cytopenia by CTCAE criteria at day 90 but did not identify any statistically significant associations (*Online Supplementary Table S9, Online Supplementary Figure S2*).

Supportive therapies

Supportive therapies administered after cilta-cel infusion are summarized in Table 4. A total of 68 patients (65%) received G-CSF support for a median duration of 23.5 days, ending at a median of 32.5 days after infusion. Neutrophil recovery occurred at a median of 16.5 days following cilta-cel infusion. In total, 40 patients (38%) received transfusion support at some time after the cilta-cel infusion, mostly within the first 30 days. Transfusions after day 30 were needed in 16 patients (15%) who required packed red blood cells and in 15 (14%) who required platelet transfusions. Eleven patients (10%) received thrombopoietin agonist support, starting at a median of 36 days after infusion and continuing for a median of 29 days. Fifty-five patients (52%) received intravenous immunoglobulins, started at a median of 67 days after infusion, and continued for a median of 75 days. Ten patients (9.5%) received a $CD34^+$ stem cell boost, administered at a median of 91 days after infusion with a median dose of $2.8x10^6$ cells/kg. All patients, except one, responded to the stem cell boost with a median time to hematologic recovery of 21 days (range, 6-96).

Infections

A total of 88 infections were reported in 51 patients (49%). Twenty-eight of the infections (32%) were severe and were reported in 15 patients (14%). The cumulative incidence of infections and infection density are shown in Figure 4. In

Table 2. Incidence and severity of cytopenias in the first 180 days following ciltacabtagene autoleucel.

A. Cytopenias by Common Terminology Criteria for Adverse Events grading.

Cytopenia	Apheresis N=105	Day -5 N=105	Day 0 N=105	Day 7 N=105	Day 14 N=103	Day 21 N=103	Day 30 N=103	Day 90 N=91	Day 180 N=49
Neutropenia, N (%)									
Any grade	31 (30)	33 (31)	71 (68)	85 (81)	69 (67)	66 (64)	69 (67)	36 (40)	21 (43)
Grade ≥3	5 (4.8)	9 (8.6)	35 (33)	62 (59)	43 (42)	30 (29)	40 (39)	12 (13)	7 (14)
Anemia, N (%)									
Any grade	59 (56)	68 (65)	81 (77)	93 (89)	82 (80)	77 (75)	73 (71)	44 (48)	18 (37)
Grade ≥3	5 (4.8)	14 (13)	19 (18)	22 (21)	10 (9.7)	9 (8.7)	11 (11)	4 (4.4)	0 (0)
Thrombocytopenia, N (%)									
Any grade	42 (40)	42 (40)	67 (64)	89 (85)	78 (76)	85 (83)	86 (83)	48 (53)	24 (49)
Grade ≥3	3 (2.9)	5 (4.8)	10 (9.5)	28 (27)	32 (31)	41 (40)	41 (40)	11 (12)	2 (4.1)
Any cytopenia, N (%)									
Any grade	88 (84)	87 (83)	104 (99)	104 (99)	98 (95)	98 (95)	97 (94)	69 (76)	35 (71)
Grade ≥3	13 (12)	20 (19)	70 (67)	72 (69)	60 (58)	56 (54)	54 (52)	22 (24)	8 (16)

B. Cytopenias by Immune Effector Cell-Associated Hematotoxicity grading.

Characteristic, N (%)	N=105	N=91
Any-grade early ICAHT	55 (52)	-
Grade ≥3 early ICAHT	11 (10)	-
Any-grade late ICAHT	-	26 (29)
Grade ≥3 late ICAHT	-	3 (3.3)

(A) Numbers and percentage of patients with any-grade and grade ≥3 neutropenia, anemia, thrombocytopenia and any cytopenia from apheresis to day 180 by Common Terminology Criteria for Adverse Events (CTCAE) grading. Any-grade cytopenias: neutropenia with absolute neutrophil count (ANC) <1,800/μL, anemia with hemoglobin <11.4 g/dL, thrombocytopenia with platelets <143,000/μL. Grade 3 cytopenias: neutropenia with ANC <1,000/μL, anemia with hemoglobin <8 g/dL, thrombocytopenia with platelets <50,000/μL. (B) Patients with any-grade and grade ≥3 cytopenia by Immune Effector Cell-Associated Hematotoxicity (ICAHT) grading. Any-grade early ICAHT: ANC ≤500/μL for <14 days between days 0-30. Grade 3 early ICAHT: ANC ≤500/μL for ≥14 days or ANC ≤100/μL for ≥7 days, between days 0-30. Any grade late ICAHT: ANC ≤1,500/μL after day 30. Grade 3 late ICAHT: ANC ≤500/μL after day 30. Evaluable patients are those who survived and had not progressed at each timepoint.

the first 30 days, bacterial infections and viral infections were equally common, with 13 cases of each reported (*Online Supplementary Table S10A*). There were five fungal infections in the study, mostly thrush (60%), all occurring within the first 30 days (Figure 4A). Between days 31 and 100, viral infections were most common (59%), and 42% of these were due to rhinovirus/enterovirus (*Online Supplementary Table S10B,* Figure 4C). After day 100, viral infections remained most common (60%), including four cases of COVID-19 (27%) and four cases of parainfluenza (27%) (*Online Supplementary Table S10C*). Respiratory infections were the most common type of bacterial infection seen throughout the study (Figure 4B).

On univariable analysis the following characteristics were associated with an increased risk of severe infections: male sex, Eastern Cooperative Oncology Group performance status ≥2 at the time of lymphodepletion (P=0.012), higher maximum grade of CRS (P=0.036), delayed neurotoxicity (P=0.025), steroid use (P=0.042), and anakinra use (P=0.024) (*Online Supplementary Table S11*). Interestingly, we did not observe any association of severe infections with markers of immunodeficiency (*Online Supplementary Table S12*) with the exception of low IgA levels at day 90 (P=0.014) being associated with a higher risk of severe infections.

Immunity

We explored the evolution of viral and pneumococcal immunity after cilta-cel by comparing changes in antibody titers between apheresis and day 90. In detail, the evolution in viral immunity of HSV-1/2 and VZV was analyzed in 24 patients with paired antibody IgG levels (*Online Supplementary Figure S3*). Of these, two patients (8%) lost immunity to HSV between apheresis and day 90, and one patient (4%) gained immunity without evidence of HSV infection (*Online Supplementary Figure S3A*). Twelve out of these 24 patients also had HSV antibody levels measured at day 180, showing loss of HSV immunity in an additional two patients (17%). Regarding VZV immunity, three of the 24 patients were excluded from analysis due to equivocal VZV antibody results at either apheresis or day 90. Among the remaining 21 patients, three (14%) lost immunity to VZV between apheresis

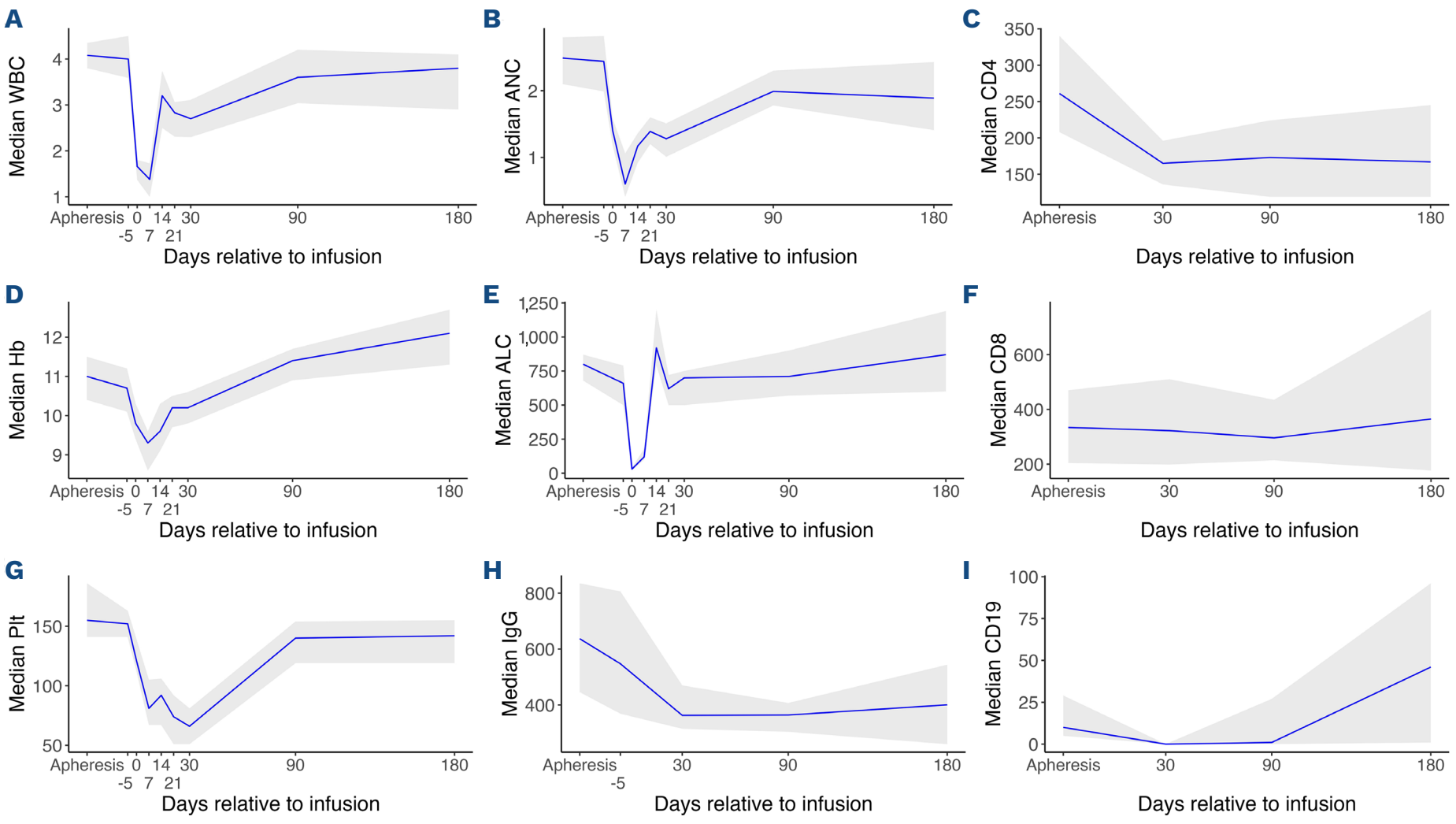

Figure 2. Cellular and humoral immune reconstitution after ciltacabtagene autoleucel. Time-series plots for cellular and humoral immune reconstitution. Solid blue lines reflect median values and gray areas reflect 95% confidence intervals. (A) Median white blood cell trend (x10^9/L) from apheresis to day 180. (B) Median absolute neutrophil count trend (x10^9/L) from apheresis to day 180. (C) Median CD4 T-cell count (cells/μL) from apheresis to day 180, based on flow cytometry. (D) Median hemoglobin trend (g/dL) from apheresis to day 180. (E) Median absolute lymphocyte count trend (cells/μL) from apheresis to day 180, based on complete blood count. (F) Median CD8 T-cell count (cells/μL) from apheresis to day 180, based on flow cytometry. (G) Median platelet count trend (x10^9/L) from apheresis to day 180. (H) Median immunoglobulin G trend (mg/dL) from apheresis to day 180. (I) Median CD19 B-cell count (cells/μL) from apheresis to day 180, based on flow cytometry. WBC: white blood cell count; ANC: absolute neutrophil count; Hb: hemoglobin; ALC: absolute lymphocyte count; Plt: platelet count; IgG: immunoglobulin G.

and day 90 (*Online Supplementary Figure S3B*). Ten of these 21 patients had VZV antibody levels measured again at day 180, showing no change in VZV immunity after day 90. The evolution in bacterial immunity to *Streptococcus pneumoniae* was analyzed in the same 24 patients based on 14 separate paired pneumococcal antibody titers between apheresis and day 90 (*Online Supplementary Figure S4*). Ten patients (42%) lost pneumococcal immunity in at least one IgG antibody titer. Four patients (17%) acquired pneumococcal immunity in at least one antibody titer. There was no proven pneumococcal infection in any of these four patients, but one patient did have unspecified bacterial bronchitis on day 54 after infusion.

Survival outcomes

Last, we compared the progression-free survival of patients based on the current grading systems for CAR T-related hematologic toxicity (*Online Supplementary Figure S5*). There was no statistically significant difference in progression-free survival when comparing patients based on the presence of grade ≥3 cytopenia (any lineage) by CTCAE criteria at day 30 or 90, early ICAHT, or late ICAHT of any grade. Only the CAR-HEMATOTOX score was prognostic, showing significantly decreased progression-free survival in patients with a high score compared to patients with a low score (P=0.0017). At the end of follow-up, 16 patients had expired, including 38% due to myeloma progression

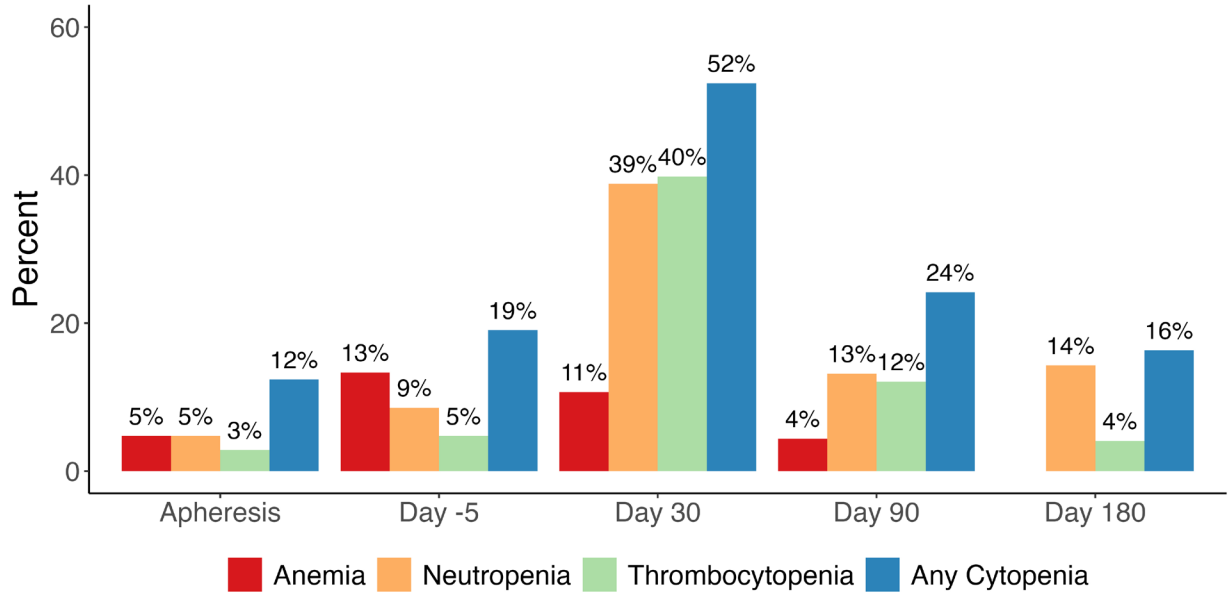

Figure 3. Cytopenias over time. Prevalence of grade ≥3 cytopenias at apheresis, day -5, day 30, day 90, and day 180. Grade 3 cytopenias: anemia with hemoglobin <8 g/dL, neutropenia with absolute neutrophil count <1,000/μL, thrombocytopenia with platelet count <50,000/μL.

Table 3. Bone marrow biopsy findings before and after (day 30, day 90 and day 180) ciltacabtagene autoleucel chimeric antigen receptor T-cell therapy.

Characteristic	Baseline N=105	Day 30 N=103	Day 90 N=91	Day 180 N=49
Marrow cellularity, N (%)				
Hypercellular	23 (23)	14 (19)	7 (10)	5 (31)
Hypocellular	16 (16)	25 (33)	22 (30)	2 (13)
Normocellular	47 (46)	29 (39)	35 (48)	8 (50)
Suboptimal	5 (4.9)	5 (6.7)	2 (2.7)	1 (6.3)
Variable	11 (11)	2 (2.7)	7 (10)	0 (0)
Unknown	3	28	18	33
Marrow cellularity, %, median (range)	35 (1-100)	30 (0.3-80)	30 (0-85)	40 (5-90)
Unknown	8	36	20	34
% CD138 positive by IHC, median (range)	7.8 (0-95)	0 (0-30)	0 (0-20)	0 (0-95)
Unknown	5	26	17	34
Fibrosis grade, median (range)	0 (0-2)	0 (0-1.5)	0 (0-1.5)	0 (0-1)
Unknown	22	43	19	34
Dysplasia present, N (%)	7 (8.5)	0 (0)	1 (1.4)	2 (13)
Unknown	23	43	19	34

Fibrosis was graded as 0 (none), 1 (mild), 2 (moderate) or 3 (marked). IHC: immunohistochemistry.

and the remainder due to non-relapse mortality (*Online Supplementary Figure S6*). The most common cause of non-relapse mortality was infection (31% of all deaths, *Online Supplementary Table S13*).

Discussion

While CAR T-cell therapy has revolutionized the treatment of RRMM, severe and/or prolonged cytopenias and risk of infection are significant toxicities with BCMA-directed CAR T cells, and lead to considerable morbidity and mortality.[7,25,26] This multicenter retrospective study provides a comprehensive characterization of the incidence, severity, and duration of cytopenias and associated infections in patients with RRMM receiving cilta-cel. We also describe risk factors associated with severe or prolonged cytopenias. In CARTITUDE-1, cytopenias were the most common treatment-related adverse event, and we observed a comparable high incidence of cytopenias in the real-world cohort.[5] Similar to the pivotal CARTITUDE-1 trial, this was a heavily pre-treated population, although 45% of patients would not have met the eligibility criteria for inclusion in the clinical trial. Severe, grade ≥3 cytopenias (per CTCAE grade) were most common in the first few weeks after cilta-cel, with two-thirds of patients experiencing severe cytopenias at day 7 and 14; this improved to about half of the patients by day 30. When evaluating individual cell lineages, the highest incidence of myelosuppression with grade ≥3 neutropenia was noted at day 7 (59%) and improved to 30-40% between days 14-30. At 3 and 6 months after cilta-cel, 13-14% of patients had grade ≥3 neutropenia with an ANC <1,000/μL. Thrombocytopenia was also common, although the incidence of grade ≥3 thrombocytopenia was lower and followed a different trajectory with a later nadir. Prior to the start of lymphodepleting chemotherapy, around 5% of patients had grade ≥3 thrombocytopenia. This increased to a third of patients at day 7 and 14 after cilta-cel, and up to 40% of patients at days 21 and 30. By 3 and 6 months after cilta-cel, only 12% and 4% of patients had grade ≥3 thrombocytopenia, respectively. Grade ≥3 anemia was less common and only seen in around 20% of patients at day 7, 10% subsequently until day 30, and less than 5% afterward. The etiology of cytopenias is multifactorial with contributions from both lymphodepleting chemotherapy and CAR T-cell-related inflammation. Indeed, we observed a bimodal trend in the trajectory of some cell lineages, with an initial dip from lymphodepletion, improvement, and subsequent worsening.[27,28]

Treatment of cytopenias included growth factor support and transfusions and the data provide expectations on supportive care and management as patients may transition back to their local oncologists after day 30. The use of G-CSF was common with about half of the patients needing G-CSF at the 1-month mark after cilta-cel, although the range was wide and some patients continued to need G-CSF for several months after CAR T-cell therapy. Thrombopoietin agonists were used in 10% of patients, with a few patients needing them for several months. The management of cytopenias often requires close collaboration between the CAR T-cell center and local oncologists. Notably, ~15% of patients required either packed red blood cells or platelet transfusions after day 30, while 10% required a stem cell boost due to persistent severe cytopenias beyond day 90. A previous real-world study described patterns of cytopenia after idecabtagene vicleucel (ide-cel), another commercial BCMA-directed CAR T-cell therapy product.[16] In parallel to our study, any grade ≥3 cytopenia was most prevalent at day 7 after ide-cel infusion; however, the observed rate was somewhat higher than the rate seen in our study (94% *vs.* 69%). At day 30, the rate of any severe cytopenias (65%)

Table 4. Supportive therapies and neutrophil recovery after ciltacabtagene autoleucel chimeric antigen receptor T-cell therapy.

Characteristic	N=105
Granulocyte colony stimulating factor, N (%)	68 (65)
First day of G-CSF, median (range)	11 (1-40)
Last day of G-CSF, median (range)	32.5 (5-309)
G-CSF duration, days, median (range)	23.5 (0-297)
CD34+ stem cell boost, N (%)	10 (9.5%)
Day of CD34+ stem cell boost, median (range)	91 (35-265)
Dose of stem cell boost, CD34+ cells x 10^6/kg, median (range)	2.8 (1.9-7.4)
Day of neutrophil recovery, median (range)	16.5 (2-330)
ANC never fell below 500 cells/uL, N (%)	12 (11.4)
Did not recover neutrophils prior to death, PD or day 100, N (%)	3 (2.9)
Transfusion after CAR T cells, N (%)	40 (38)
Packed RBC transfusion within 30 days	33 (31)
Platelet transfusion within 30 days	23 (22)
Packed RBC transfusion >30 days	16 (15)
Platelet transfusion >30 days	15 (14)
Thrombopoietin agonist, N (%)	11 (10)
First day of TPO agonist, median (range)	36 (23-77)
Last day of TPO agonist, median (range)	65 (30-309)
TPO agonist duration, days, median (range)	29 (3-258)
Intravenous immunoglobulins, N (%)	55 (52)
First day of IVIG, median (range)	67 (12-225)
Last day of IVIG, median (range)	142.5 (12-463)
IVIG duration, days, median (range)	75 (0-381)

When the exact day of supportive therapy initiation or discontinuation was unknown due to administration outside of the institution, day of first or last documentation of use in the medical record was used as a surrogate. Neutrophil recovery was defined as first day of absolute neutrophil count (ANC) greater than 500 cells/μL sustained on two consecutive checks without granulocyte colony-stimulating factor use in the preceding 7 days. Median and range of neutrophil recovery reflects data for the 90 patients whose ANC fell below 500 cells/μL after ciltacabtagene autoleucel and recovered in the absence of death or progression. G-CSF: granulocyte colony-stimulating factor; PD: progressive disease; CAR: chimeric antigen receptor; RBC: red blood cell; TPO: thrombopoietin; IVIG: intravenous immunoglobulins.

was still elevated; the rate of severe neutropenia (39%) was similar to that seen with standard-of-care cilta-cel, however, rates of severe anemia (29%) and severe thrombocytopenia (51%) were numerically higher compared to those seen with cilta-cel. Eventually, by day 90, severe cytopenias improved in the majority of the patients, as seen in our study.

We identified subgroups of patients at higher risk of grade ≥3 cytopenias at day 30 and 90, although given our small sample size, the power to detect such an association was limited. Extramedullary disease, high-risk cytogenetics, more than four prior therapies, baseline cytopenias, high CAR-HEMATOTOX score, and tocilizumab use were associated with a higher risk of severe cytopenias at day 30. At day 90, risk factors included more heavily pre-treated disease and baseline thrombocytopenia at apheresis. Notably, neither bridging therapy nor the use of alkylators as bridging therapy was associated with grade ≥3 cytopenia.

Infections are common after CAR T-cell therapy and constitute the most common cause of non-relapse mortality, which is consistent with our observed data.[7,29-31] Two previous studies have described infectious complications with BCMA-directed CAR T-cell therapies. The first was a single-center analysis that included 55 patients on clinical trials, of whom 55% received ide-cel, 15% cilta-cel, and 31% an experimental product.[32] In total, 47 infections

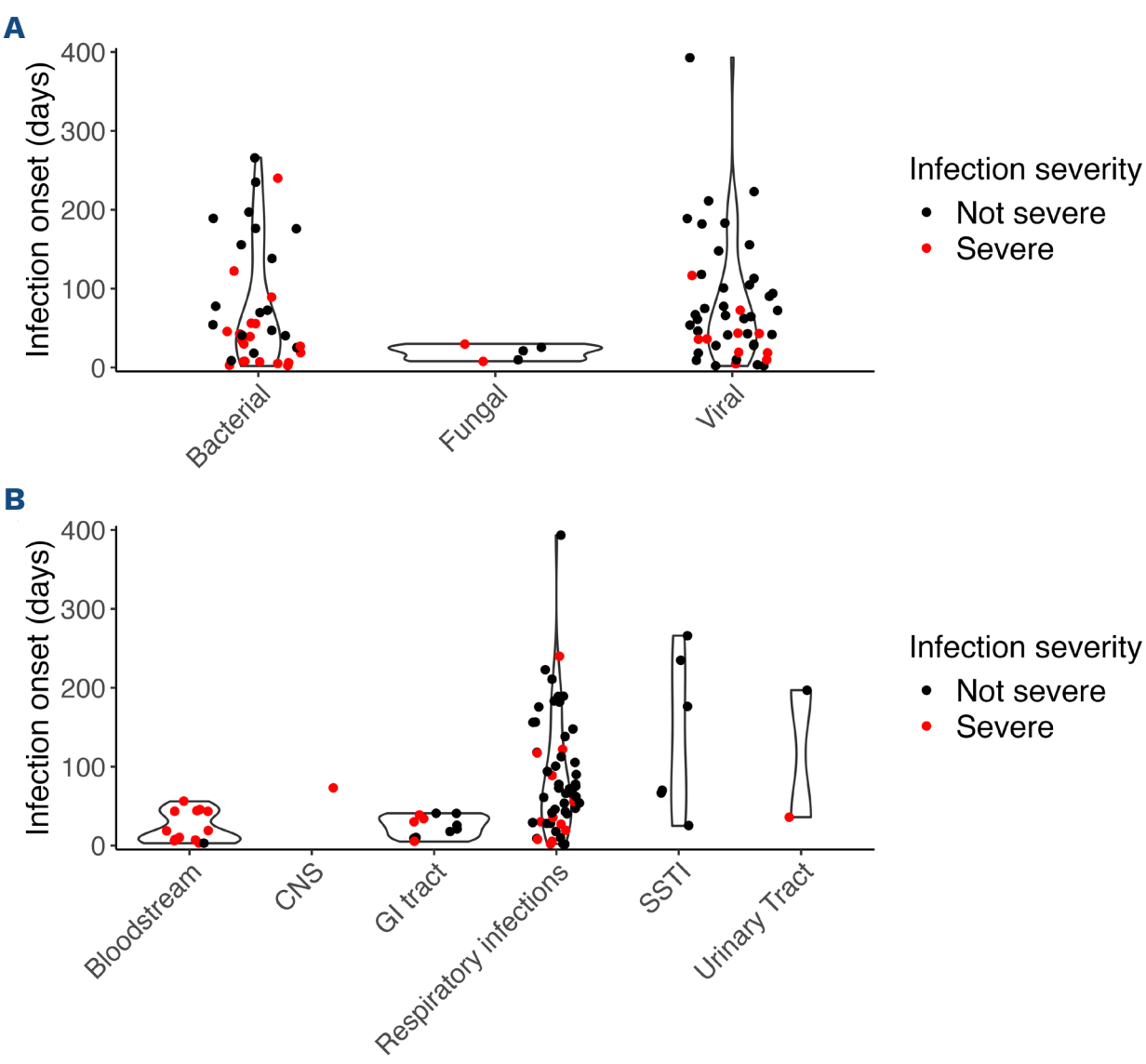

Figure 4. Cumulative incidence of infection and infection density following ciltacabtagene autoleucel infusion. (A) Cumulative incidence of infection by type of infection (bacterial, fungal, and viral) and severity. (B) Cumulative incidence of bacterial infections by type and severity. (C) Cumulative incidence of viral infections by type and severity. CNS: central nervous system; GI: gastrointestinal; SSTI: skin and soft tissue infections; CMV: cytomegalovirus; COVID-19: coronavirus disease 2019; JC virus: human polyomavirus 2; RSV: respiratory syncytial virus; VZV: varicella-zoster virus (shingles).

were reported in 29 patients (53%), with 53% of the infections occurring within the first 100 days after the infusion (40% bacterial, 53% viral, 92% mild-moderate, and 68% involving the respiratory tract).[32] A multicenter study of 52 patients who received standard-of-care ide-cel reported infectious complications until day 100 after infusion. Infections occurred in 54% of patients and were grade ≥3 in 23%.[16] Earlier infections in the first 30 days were typically bacterial (68%) and severe (50%). Later infections between days 31 and 100 were 50% bacterial and 42% viral; only 13% were grade ≥3. Longer time from last bridging treatment to lymphodepletion was the only significant risk factor for infection.[16]

When comparing our data on infections with the data from the studies mentioned above, we had a similar proportion of patients who experienced an infection following CAR T-cell therapy (49% in our study *vs*. 54% and 53% in the afore-mentioned studies). In addition, while the number of patients with severe infections was lower with standard-of-care cilta-cel compared to standard-of-care ide-cel (14% *vs*. 23%, respectively), the number of infections of any grade within 100 days of CAR T-cell infusion was higher with standard-of-care cilta-cel (62 *vs*. 46), despite antibiotic prophylaxis.[16] With standard-of-care ide-cel, early infections in the first 30 days were typically bacterial (68%), and later infections between days 31-100 were more evenly distributed between bacterial and viral causes, as cited above. In contrast, with standard-of-care cilta-cel, early infections were evenly distributed between bacterial (42%) and viral (42%) causes, while later infections were mostly viral (~60%).[16] When comparing our data on infections with those from CARTITUDE-1, the proportion of patients who developed an infection (49% *vs*. 58% on trial) or severe infection was greater in the trial (14% *vs*. 20% on trial).[5] Direct comparisons of infectious complications are limited by the fact that our knowledge and infectious disease prophylaxis have rapidly evolved in a short time period from the approval of ide-cel to cilta-cel, with more widespread use of intravenous immunoglobulins and other prophylactic measures.

Most institutions in the USA and internationally have adopted post-autologous hematopoietic cell transplant revaccination practices after administration of CAR T-cell therapy; however, data on post-CAR T-cell immunity are very limited.[33] A prior study with standard-of-care ide-cel found evidence that immunity for HSV, VZV, CMV, and pneumococcus is lost in a proportion of patients but not universally.[16] Likewise, we observed that immunity for HSV, VZV, and pneumococcus is lost in a proportion of patients after cilta-cel. Notably, vaccine immunity loss was less common following CD19 CAR T-cell therapy, which may contrast with BCMA CAR T cells.[28] Given these findings, revaccination may be particularly beneficial after cilta-cel, especially for pneumococcus. Future studies with longer follow-up and larger cohorts of patients are necessary to assess antibody titers over time for all commonly used vaccines.

The limitations of our study include its relatively small sample size and retrospective nature. Longer follow-up and a larger number of patients are needed to fully elucidate the risk factors and longer-term patterns of hematologic and infectious complications after administration of commercial cilta-cel. Furthermore, daily post-infusion ANC data were not available; thus, categorization into clinical phenotypes of neutrophil recovery was not possible.[34] Prospective studies will be important to provide insights into more standardized use of supportive care measures, including common strategies for antimicrobial prophylaxis as well as the use of G-CSF, stem cell boosts, and intravenous immunoglobulins.

In conclusion, cytopenias and infections are common complications following the administration of standard-of-care cilta-cel. A significant proportion of deaths were attributed to non-relapse mortality, particularly infections, highlighting the critical need for enhanced supportive care strategies. As cilta-cel is increasingly used in earlier lines of therapy, optimizing supportive care protocols such as growth factor support, intravenous immunoglobulins, and infection prophylaxis will be paramount for improving patients' outcomes.

Disclosures

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Contributions

DD, JML and DKH wrote the first draft of the manuscript. LCP and CMCL analyzed the data. All authors contributed patients to this analysis, provided critical feedback, edited and wrote the manuscript, and approved the final manuscript.

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Data-sharing statement

For original deidentified data that underlie the reported results, please contact Doris.Hansen@moffitt.org.

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