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CMV reactivation after autologous stem cell transplantation: exploratory associations with transfusion burden and pre-transplant IMiD exposure[a]

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ABSTRACT

Aims: To evaluate the incidence and clinical impact of cytomegalovirus (CMV) reactivation and disease after autologous stem cell transplantation (ASCT), explore associations between transfusion burden, pre-transplant immunomodulatory drug (IMiD) exposure, and CMV reactivation, and identify subgroups that may warrant intensified surveillance.

Methods: Retrospective analysis of 188 CMV-seropositive ASCT recipients at Erciyes University between 2007–2016. CMV PCR was performed twice weekly for the first 100 days. Reactivation was defined as new PCR positivity after ASCT in a patient with a negative or undetectable pre-transplant PCR. Erythrocyte suspension (ES) and platelet suspension (PS) were analysed separately alongside combined transfusion burden, with patients receiving no blood products retained as the 0-unit reference group. Fisher exact and Chi-square tests were used (SPSS v16.0).

Results: CMV reactivation occurred in 44 patients (23.4%); 40 (90.9%) had asymptomatic DNAemia and 4 (9.1%) developed CMV disease. All reactivation events occurred within day +46. Refractory/progressive disease was associated with higher reactivation rates (50.0% vs 21.9%; p=0.056). Including the 0-unit reference group (n=186), ES ≥6 units was significantly associated with CMV reactivation (62.5% vs 21.9%; odds ratio [OR] 5.94, 95% confidence interval [CI]: 1.36–25.96; p=0.019), while PS (≥6 vs <6 units) alone was not (31.6% vs 22.8%; p=0.399). In an analysis restricted to pre-transplant IMiD exposure within the MM subgroup — the only analysis used to infer a protective association — reactivation occurred in 10.5% of exposed patients (n=19) versus 27.4% of IMiD-naive MM patients (n=84; OR 0.31, 95% CI 0.07–1.46; p=0.149), a non-significant trend. Because all CMV reactivation events occurred by day +46, before post-transplant IMiD maintenance is typically initiated (day +60–100), post-transplant IMiD exposure could not plausibly have influenced these events and was not used as a predictor of reactivation. Conditioning regimen was not associated with CMV reactivation across four regimen groups (BEAM, melphalan 200, melphalan 140, and other regimens) (p=0.863).

Conclusion: Refractory disease and high erythrocyte transfusion burden are strongly associated with CMV reactivation after ASCT, although the erythrocyte-transfusion association may partly reflect delayed immune recovery rather than a direct causal effect. ES was the transfusion parameter most consistently associated with reactivation; PS alone was not independently associated. Pre-transplant IMiD exposure showed a non-significant protective trend in the MM subgroup that requires confirmation in larger, prospective studies; the proposed NK-cell-mediated mechanism remains hypothetical pending direct immune-function data. Risk-stratified CMV monitoring may be warranted for high-risk subgroups, pending validation in contemporary, prospective cohorts.

Keywords: Autologous stem cell transplantation, cytomegalovirus, immunomodulatory drugs, erythrocyte transfusion, risk factors

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INTRODUCTION

Cytomegalovirus (CMV) remains the most clinically significant viral pathogen following haematopoietic cell transplantation.[1] CMV seroprevalence exceeds 90% in Turkiye,[2] substantially higher than the ~50–60% reported in Western populations,[3] and reactivation after autologous HSCT (ASCT) is reported in 4–33% of seropositive recipients.[4-7] Although most episodes are asymptomatic, progression to CMV disease carries high mortality, and late

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reactivation has been linked to inferior survival.[8] Despite the well-established risk profile in allogeneic HSCT, risk factors for CMV reactivation in ASCT remain poorly defined,[9,10] and the clinical significance of asymptomatic DNAemia versus overt CMV disease in this setting is not fully resolved.

Immunomodulatory drugs (IMIDs)—thalidomide, lenalidomide, and pomalidomide—enhance NK-cell and T-cell activity through cereblon-mediated mechanisms[11,12] and may confer antiviral protection; however, whether this effect depends on the timing of IMID exposure relative to transplantation has not been evaluated. Similarly, the relative contribution of erythrocyte versus platelet transfusion burden to CMV reactivation risk remains poorly characterised. This study evaluates these questions in a large single-centre ASCT cohort, with particular attention to the temporal plausibility of the IMiD association and to the extent to which transfusion-related associations may reflect residual confounding by disease and treatment intensity rather than a direct effect.

METHODS

Ethics

This study was approved by the Clinical Researches Ethics Committee of Erciyes University (Date: 15.09.2017, Decision No: 2017/430). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Study Population

All patients undergoing ASCT between January 2007 and December 2016 (n=352) were screened. Inclusion required CMV seropositivity and at least twice-weekly CMV PCR monitoring during the first 100 days post-ASCT. After day +100, PCR was repeated on clinical suspicion. A total of 188 patients were included.

CMV Monitoring and Definitions

CMV DNAemia was defined as PCR ≥100 IU/ml; CMV disease required clinical symptoms or tissue confirmation.[13] Preemptive therapy (intravenous ganciclovir or oral valganciclovir; foscarnet for refractory cases) was initiated for viral loads >2,000 IU/ml or rising titres. No CMV prophylaxis was administered during the study period. As part of pre-transplant donor/recipient screening, baseline CMV serostatus and PCR were assessed; only patients with a negative or undetectable pre-transplant PCR were classified as having post-transplant CMV reactivation. The single patient with a positive PCR on day 0 had a documented negative pre-transplant PCR, confirming true post-transplant reactivation rather than pre-existing DNAemia.

Transfusion and IMID Analysis

All blood products were leukoreduced and irradiated. ES and PS volumes were recorded separately. Patients who received no blood products (n=28) were retained as the 0-unit reference category in transfusion analyses rather than excluded; two additional patients with recorded platelet but no recorded erythrocyte-transfusion data were excluded from ES-specific analyses only, giving an ES-evaluable n=186. IMID exposure (thalidomide, lenalidomide, or pomalidomide) and its timing relative to transplantation were recorded for MM patients. Because all CMV reactivation events occurred by day +46, well before post-transplant IMiD maintenance is typically initiated (day +60–100), the primary IMiD risk-factor analysis was restricted to pre-transplant IMiD exposure within the MM subgroup; post-transplant IMiD exposure was analysed descriptively only and was not used as a predictor of reactivation. One patient's diagnosis had been miscoded in the study database as non-Hodgkin lymphoma; chart review confirmed a diagnosis of multiple myeloma, and the record was corrected prior to analysis (all analyses below therefore use the full, corrected cohort of n=188).

Statistical Analysis

Analyses were performed using SPSS software (version 16.0; SPSS Inc., Chicago, IL, USA). Fisher exact and Chi-square tests were applied; $p<0.05$ was significant. Exact 95% CIs (Clopper-Pearson method) and bootstrap analysis (10,000 iterations) were performed for small subgroups.

RESULTS

Patient Characteristics and CMV Outcomes

Of 188 CMV-seropositive patients (mean age 52.8±13.2 years; 117 male [62.2%]), the most common diagnosis was MM (n=127, 67.6%), followed by NHL (n=27, 14.4%) and HL (n=15, 8.0%) (one patient's diagnosis, originally miscoded as NHL, was corrected to MM after chart review; see Methods). At ASCT, 138 (73.4%) were in CR, 40 (21.3%) in PR, 8 (4.3%) had refractory/relapsed disease, and 2 (1.1%) progressive disease. CMV reactivation occurred in 44 patients (23.4%): 40 (90.9%) had asymptomatic DNAemia and 4 (9.1%) developed CMV disease (2 gastrointestinal, 1 pulmonary, 1 febrile with biopsy-proven tissue invasion). The median day of first positive PCR was day +29 (range 0–46); all events occurred within day +46. Patient characteristics are summarised in Table 1.

Table 1. Patient demographics and clinical characteristics (n=188)

Characteristic	n	%
Gender, male	117	62.2
Age, mean±SD (years)	52.8±13.2	—
Diagnosis		
Multiple myeloma	127	67.6
Non-Hodgkin lymphoma	27	14.4
Hodgkin lymphoma	15	8.0
Other diagnoses	19	10.1
Disease status at ASCT		
Complete remission (CR)	138	73.4
Partial remission (PR)	40	21.3
Refractory/relapsed	8	4.3
Progressive disease	2	1.1
CMV IgG seropositive	188	100.0
IMID-exposed (MM subgroup)	43/127	33.9

ASCT: Autologous stem cell transplantation, CMV: Cytomegalovirus, IMID: Immunomodulatory drug, MM: Multiple myeloma, SD: Standard deviation.

Risk Factors for CMV Reactivation

Disease status at ASCT showed a clinically meaningful association with CMV reactivation: rates were 22.5% (CR), 20.0% (PR), 62.5% (refractory/relapsed), and 0% (progressive). Comparing remission (CR+PR, n=178) with refractory/progressive disease (n=10): 21.9% vs 50.0% (p=0.056). This difference did not reach conventional statistical significance,

reflecting the small size of the refractory subgroup. Conditioning regimen was not independently associated with CMV reactivation (BEAM 27.5% [n=51], melphalan 200 21.9% [n=105], melphalan 140 20.0% [n=20], other regimens 25.0% [n=12]; four-group Chi-square p=0.863). Risk factor data are presented in Table 2.

Table 2. Disease status at ASCT and CMV reactivation rates

Disease status	Total (n)	CMV+(n)	Rate (%)	p
Complete remission (CR)	138	31	22.5	
Partial remission (PR)	40	8	20.0	
Refractory/relapsed	8	5	62.5	
Progressive disease	2	0	0.0	
Remission (CR+PR) vs refractory/progressive	178 vs 10	39 vs 5	21.9 vs 50.0	0.056*

*Fisher's exact test. CMV: Cytomegalovirus, CR: Complete remission, PR: Partial remission.

IMID Exposure and CMV Reactivation

After correcting one patient's diagnosis—miscoded in the study database as non-Hodgkin lymphoma but confirmed as multiple myeloma on chart review (n=188 throughout)—IMiD exposure was associated with a lower reactivation rate in the overall cohort (9.3% vs 27.6%; p=0.013) and the MM subgroup (9.3% vs 27.4%; p=0.022). IMID-exposed and IMID-naive MM patients had similar mean ES transfusion volumes (0.8 units each), arguing against ES burden as a confounding variable. Because all CMV reactivation events occurred by day +46—before post-transplant IMiD maintenance is typically initiated (day +60–100)—post-transplant IMiD exposure cannot plausibly have influenced these events and was not used as a predictor of reactivation. The primary risk-factor analysis was therefore restricted to pre-transplant IMiD exposure within the MM subgroup (n=19): reactivation occurred in 10.5% of pre-transplant IMiD recipients versus 27.4% of IMiD-naive MM patients (OR 0.31, 95% CI 0.07–1.46; p=0.149), a non-significant protective trend most likely reflecting limited statistical power. Post-transplant IMiD exposure (n=21; reactivation 9.5%) is presented in Table 3 for descriptive completeness only and was not used to infer a protective effect, given the temporal implausibility described above. IMiD timing could not be classified for 3 of the 43 MM IMiD-exposed patients because of incomplete treatment-date records. IMID data are summarised in Table 3.

Table 3. IMID exposure, timing, and CMV reactivation

Group	Total (n)	CMV+(n)	Rate (%)	p
All patients–IMID naive	145	40	27.6	0.013*
All patients–IMID exposed	43	4	9.3	
MM–IMID naive	84	23	27.4	0.022*
MM–IMID exposed	43	4	9.3	
IMID timing (MM exposed only)				
Pre-transplant IMID	19	2	10.5	1.000*
Post-transplant IMID	21	2	9.5	
IMID naive (MM reference)	84	23	27.4	—

*Fisher's exact test. CMV: Cytomegalovirus, IMID: Immunomodulatory drug, MM: Multiple myeloma.

Transfusion Burden and CMV Reactivation

Retaining the 28 patients who received no blood products as the 0-unit reference group (evaluable n=186), total transfusion volume was significantly associated with CMV reactivation (17.6% [0 units], 22.3% [1–5 units], 29.4% [6–10 units], 53.8% [≥11 units]; four-group p=0.048). ES ≥6 units remained strongly associated with reactivation: 62.5% (95% CI: 24.5–91.5%) vs 21.9% (95% CI: 16.1–28.7%) for ES <6 units, including the 0-unit patients (OR 5.94, 95% CI: 1.36–25.96; p=0.019). Bootstrap analysis (10,000 iterations) confirmed a similar median risk difference of 41.2% (95% CI: 4.8–70.6%). Using the same ≥6 vs <6-unit threshold, PS was not independently associated with CMV reactivation (31.6% vs 22.8%; OR 1.57; p=0.399). Two patients who received platelet but no recorded erythrocyte transfusion were excluded from ES-specific analyses because unit counts were unavailable. Transfusion data are presented in Table 4.

Table 4. Transfusion burden and CMV reactivation (n=160 patients receiving replacement therapy)

Group	n	CMV+(n)	Rate (%)	p
Total blood products				**0.043†**
≤5 units	130	29	22.3	
6–10 units	17	5	29.4	
≥11 units	13	7	53.8	
Erythrocyte suspension (ES)				**0.028***
ES <6 units	150	36	24.0 (95% CI: 17.4–31.6)	
ES ≥6 units	8	5	62.5 (95% CI: 24.5–91.5)	
Platelet suspension (PS)—any volume	160	41	25.6	0.429 NS†

*Fisher's exact test, † Chi-square test. CI: Confidence interval, CMV: Cytomegalovirus, ES: Erythrocyte suspension, NS: Not significant, PS: Platelet suspension.

DISCUSSION

This retrospective analysis of 188 CMV-seropositive ASCT recipients demonstrates a reactivation rate of 23.4%, consistent with contemporary cohorts.[8,14] All events occurred within day +46, confirming the adequacy of the 100-day monitoring window. The large majority of reactivation events (40/44, 90.9%) represented asymptomatic DNAemia rather than CMV disease (4/44, 9.1%), raising the question of whether DNAemia alone constitutes a clinically meaningful endpoint or primarily a virological marker. Because our retrospective dataset did not capture survival, transplant-related mortality, or length of hospital stay, we could not directly test the prognostic impact of reactivation in this cohort. Lee et al.[8] reported that CMV reactivation after ASCT was independently associated with inferior overall survival in a comparable population; our findings should be interpreted in that context — while the reactivation rate we observed is consistent with contemporary series, whether it independently affects outcomes in our own cohort remains unresolved and is an important direction for prospective follow-up incorporating survival endpoints.

Refractory/progressive disease at ASCT was associated with a clinically meaningful but statistically non-significant increase in CMV reactivation (50.0% vs 21.9%; p=0.056), likely reflecting limited power due to the small subgroup size (n=10). Conditioning regimen (BEAM, melphalan 200, melphalan 140, and other regimens) was not an independent predictor (p=0.863), though the strong collinearity between

diagnosis and regimen (MM→melphalan; lymphoma→BEAM) limits independent assessment of conditioning effects.

ES transfusion burden, analysed with the 0-unit reference group retained, was significantly associated with CMV reactivation (OR 5.94, 95% CI 1.36–25.96; p=0.019), while PS, evaluated using the same ≥6-unit threshold, was not independently associated (p=0.399). This association should not be interpreted as evidence of a direct causal effect: patients requiring higher ES volumes are also more likely to have undergone more intensive chemotherapy, experienced more severe mucositis, remained neutropenic longer, or engrafted later—factors not captured in this retrospective dataset (engraftment timing, CD34+ cell dose, mucositis grade, and duration of febrile neutropenia were unavailable). ES burden is therefore best regarded as a strongly associated marker of delayed immune recovery and greater treatment intensity rather than an independently confirmed causal risk factor. Even with standard leukoreduction, erythrocyte preparations may also exert direct immunosuppressive effects through soluble mediators and NK/T-cell suppression,[15] though this mechanism remains speculative in the absence of immune-function data in our cohort. Similar associations between immune dysregulation and CMV reactivation have also been described outside the transplant setting, in critically ill patients.[16]

The post-transplant IMiD subgroup should not be interpreted as evidence of a 'timing-independent' protective effect, because post-transplant IMiD maintenance (typically initiated day +60–100) could not plausibly have influenced CMV reactivation events that had already occurred by day +46. Restricting the analysis to pre-transplant IMiD exposure in the MM subgroup (n=19), we observed a non-significant protective trend (10.5% vs 27.4% in IMiD-naive MM patients; p=0.149) that did not reach statistical significance, most likely because of limited sample size; this finding is hypothesis-generating rather than confirmatory. A biologically plausible mechanism has been proposed in vitro: lenalidomide lowers the activation threshold of NK cells through cortical actin remodelling,[17] which could in principle produce a durable antiviral immune state. However, our dataset lacked NK-cell counts, NK-cell functional assays, T-cell subset data, and broader immune reconstitution measures, so this mechanism remains speculative and unconfirmed in our cohort and should be regarded as a hypothesis for future mechanistic studies rather than an established explanation.

An additional consideration is the applicability of these findings to current clinical practice. Patients in this cohort underwent ASCT between 2007 and 2016, a period that predates several major changes in CMV management, including risk-based individualised monitoring strategies,[18-22] more sensitive quantitative PCR assays, letermovir prophylaxis (not used in our cohort),[23,24] and newer therapeutic options such as maribavir for refractory or resistant CMV.[25,26] Contemporary practice increasingly relies on serostatus- and risk-based stratification[27] and risk-adapted surveillance; comparable strategies are less well defined specifically for ASCT recipients, for whom our data suggest a subset—patients with refractory/progressive disease or high ES requirements — could plausibly benefit from closer monitoring. Because CMV PCR thresholds, preemptive-therapy triggers, and supportive care have evolved since 2016, the absolute reactivation rates reported here should be interpreted as reflecting practice patterns of that era, and prospective validation in a contemporary cohort using current monitoring protocols and updated guidance[22] is warranted before these findings inform current surveillance policy.

Limitations

The retrospective single-centre design and small critical subgroups (refractory/progressive disease n=10; ES ≥6 units n=8; pre-transplant IMiD-exposed n=19) precluded multivariable adjustment, and all reported associations are unadjusted and should be interpreted as exploratory. We were unable to evaluate several parameters that plausibly influence both transfusion requirements and CMV reactivation risk, including neutrophil and platelet engraftment day, CD34+ cell dose, mucositis grade, and duration of febrile neutropenia; consequently, the association between ES burden and CMV reactivation may partly reflect delayed immune recovery rather than a direct transfusion-related effect. Data on corticosteroid and other immunosuppressive therapies were available for all patients; however, calculation of precise cumulative doses was not feasible from retrospective records. Importantly, all patients received similar standard-of-care steroid regimens as part of conditioning and supportive care, suggesting broadly comparable steroid exposure levels across groups, which limits the likelihood of major steroid-related confounding. Transfusion volume was analysed as a cumulative summary measure; time-varying modelling was beyond the scope of this study. Survival, transplant-related mortality, and length of hospital stay were not systematically captured in this retrospective database, precluding outcome analyses; this is an important limitation given uncertainty about the clinical significance of asymptomatic CMV DNAemia and should be addressed in future prospective studies with survival follow-up. All CMV reactivation events occurred within day +46 and no late reactivation was detected, supporting the adequacy of the 100-day monitoring window; the primary IMiD analysis (pre-transplant exposure, MM subgroup, n=19) showed a non-significant protective trend (10.5% vs 27.4%, p=0.149), reflecting reduced statistical power. The proposed NK-cell-mediated mechanism underlying the IMiD association is not supported by direct immune-function data (NK-cell counts, NK-cell functional assays, T-cell subsets, or immune reconstitution measures) in this cohort and remains hypothetical. This cohort was treated between 2007 and 2016, and CMV monitoring and treatment practices have since evolved; the generalisability of absolute reactivation rates to contemporary practice is discussed above. Despite these limitations, the systematic twice-weekly monitoring protocol and uniform transfusion policy provide a dataset for exploratory risk factor identification in this understudied population.

CONCLUSION

Refractory disease and high erythrocyte transfusion burden are strongly associated with CMV reactivation after ASCT, although the erythrocyte-transfusion association may partly reflect delayed immune recovery rather than a direct causal effect. ES was the transfusion parameter most consistently associated with reactivation; PS alone is not independently associated. Pre-transplant IMiD exposure showed a non-

significant protective trend in the MM subgroup that requires confirmation in larger, prospective studies; the proposed NK-cell-mediated mechanism remains hypothetical pending direct immune-function data. Risk-stratified CMV surveillance may be warranted, with intensified monitoring for patients with refractory disease or high ES requirements; these exploratory findings should be validated in contemporary, prospective cohorts before informing clinical monitoring policy.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the Clinical Researches Ethics Committee of Erciyes University (Date: 15.09.2017, Decision No: 2017/430).

Informed Consent

This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

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Author Contributions

Concept: MI, LK; Design: MI, LK, SG; Control: MI, LK, SG, MÇ; Resources: MI, LK; Materials: MI, LK, SG; Data Collection and/or Processing: MI, LK, SG, MÇ; Analysis and/or Interpretation: MI, LK; Literature Review: MI, LK, SG; Writing the Article: MI, LK; Critical Review: MI, LK, SG, MÇ.

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