MSD report on the 52nd Annual Meeting of the EBMT

22 - 25 March 2026

IFEMA Madrid, Spain

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Source: www.ebmt.org/annual-meeting-2026

Content

Preface

The 52nd Annual Meeting of the EBMT took place at IFEMA in Madrid from 22 to 25 March 2026. For the sixth consecutive year we present a focused summary of the meeting's cytomegalovirus (CMV) related contributions, covering posters, oral sessions and satellite symposia that address prevention, diagnosis, antiviral therapy and immune-based strategies in the hematopoietic stem cell transplantation setting.

This year's programme again demonstrated that CMV remains 'the troll in transplantation' after allogeneic HSCT. But also that preventive and therapeutic options continue to evolve. The program reflected a wide range of clinical and translational work aimed at improving management. Several presentations examined real-world experience and trial data related to letermovir prophylaxis, including effects on CMV incidence, timing of administration, and observations about immune recovery. Other sessions reported on the use of maribavir for difficult-to-treat or resistant CMV infections, with analyses of efficacy, safety and practical considerations in varied transplant populations, including CAR T recipients.

The meeting also gave updates on the most recent advances, such as adoptive CMV-specific T-cell therapies, diagnostic advances, and case reports that highlight complex clinical decision making.

We thank the investigators, clinicians and allied health professionals who shared their work in Madrid. Their work keeps improving understanding of CMV biology, prevention and treatment after transplantation. Last but not least, we also thank the organization for making the congress possible, and for their support to this report.

We hope this report, which includes almost all* CMV topics presented at EBMT 2026, again will be a practical reference for hematologists, infectious disease specialists and transplant teams.

Enjoy reading!

* Off-label information on letermovir is excluded

Takeda Industry Symposium IS09
March 22nd, 2026

Illuminated Constellations: How Diverse Experiences Guide Practical CMV Management | Takeda

Effectively Identifying and Treating Patients with Cytomegalovirus: A Panel Discussion

Rafael Duarte (Madrid, Spain), Johan Maertens (Leuven, Belgium), Eva Wagner-Drouet (Mainz, Germany)

The session was opened by Dr. Duarte, this year's congress president. He introduced the speakers Prof. Maertens from Belgium and Dr. Wagner-Drouet from Germany.

The symposium was organized around five questions:

1. What is the value of early identification of CMV infection in post-transplant HCT recipients?
2. How do you incorporate viral load monitoring into your practical management of CMV infection?
3. In what ways have newer anti-CMV agents influenced your approach to the early identification and intervention of CMV infection?
4. What factors influence your clinical decision-making when deciding on an appropriate duration of treatment with maribavir?
5. Does the risk of resistance to current antivirals affect your treatment decisions for CMV?

1. What is the value of early identification of CMV infection in post-transplant HCT recipients.
The session kicked off with a poll asking the attendees how they currently manage CMV. The majority of colleagues responded that they make use of both prophylaxis and pre-emptive therapy. More than 70% of the attendance have access to letermovir prophylaxis. The speakers discussed the last years ECIL-10 guideline.[1] Letermovir should be started as early as possible. And even though blips can occur, it should be continued to at least 100 days post-transplant or up to 200 days when applicable in patients who are at high risk of CMV disease.

There is no scientific consensus which viral load cut-off should be used to start to pre-emptive therapy. The HCPs discussed decisions need to be made on a case-by-case basis. The pivotal trials of letermovir clearly showed a significant decrease of CMV infection and mortality. The original study by Marty et al. has been criticized by not including every patient group at the time.[2] Though the meta-analysis published a few years ago by the Chemaly group[3] confirmed the consistent reduction in CMV-reactivation across different patient groups. A post-hoc mortality analysis of Ljungman et al.[4] of the multicenter, double-blind, placebo-controlled phase 3 trial by Marty (n=570) also found a positive effect on mortality outcomes; there was a significant difference (p=0,03) in mortality between participants with cs CMVi who had received letermovir prophylaxis and participants who received placebo (receiving pre-emptive therapy after cmv-reactivation).

Kaplan-Meier Plot of Overall Survival by 6 Weeks Among Day 100 Survivors with 1st Late CMV disease in Entire Cohort (N = 177)

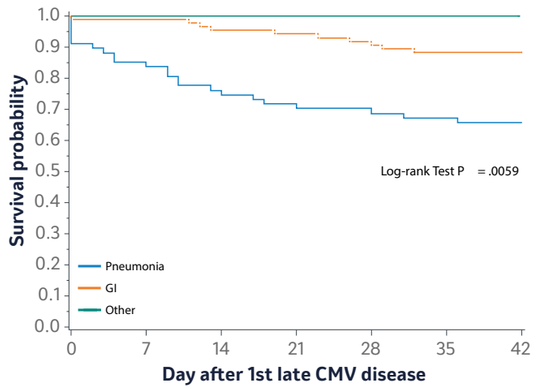

Figure 1: Adapted from Sadowska-Klasa, A et al. 2024

However, there is a group of patients (31 of 68 patients, 46%) that remains with developed csCMVi after discontinuation of letermovir, as described by Putz et al.[5] A growing amount of evidence shows delayed humoral immunity is associated with late onset CMV infections, after cessation of letermovir prophylaxis. When ceasing prophylaxis the risk of late CMV disease and mortality remains.[5,6,7,8]
Dr. Wagner-Drouet said "the younger generation of HCPs does not even see CMV as a real threat anymore, due to the introduction of letermovir." She put additional focus on the graph showing the 6-week survival after late-CMV diagnosis (Figure 1), to highlight the impact of organ involvement in late infections. Especially lung involvement leads to a worse outcome of disease.

A recent publication of Takenaka et al, shows that late onset CMV infection was associated with a lower overall survival.[9] This retrospective cohort analysis evaluated the impact of letermovir prophylaxis on posttransplant outcomes using the registry database of the Japanese Society for Transplantation and Cellular Therapy, and included 6004 adult patients who underwent a-HSCT between 2017 and 2019. Letermovir prophylaxis was administered to 1640 patients.

Similarly, the analysis of Olivari et al, a single-center, retrospective observational study in 236 CMV-seropositive aHSCT patients investigating the risk factors associated with an increased risk of multiple CMV reactivations or the development of CMV disease, showed 84% (p=0,046) of patients 1 year overall survival, and 75% (p=0,36) of 1 year progression-free survival. Both 1-year overall survival and progression-free survival decreased to 67% in patients with more than two reactivations.10 The conclusion was that, while letermovir is effective at preventing csCMVi, patients remain at risk for breakthrough infections and late-onset CMV disease. The speakers also concluded that breakthrough and late-onset CMV infections with letermovir are associated with poor survival.

2. How do you incorporate viral load monitoring into your practical management of CMV infection?
Common practice among the attendees is to monitor CMV once- (61%) or twice (33%) weekly both during and after prophylaxis (89%).
The speakers discussed the ECIL-10 guideline on monitoring.1 'Since in daily practice breakthrough infections are seen in approx. 10%, may be even 20% of patients' Maertens said, 'it remains of high importance.' Also, the cause of an infection can be multi-factorial. So especially in patients with an increased risk, there should be extended

monitoring in place. Dr. Wagner-Drouet re-emphasized also the patient's individual dynamics.

The audience reflected how they can incorporate monitoring in daily response and the speakers discussed if it can be used to decide duration of prophylaxis. Wagner-Drouet: "It can guide the recommendation for withholding therapy for low level viremia, secondary prophylaxis, or additional treatment." In Germany, they implemented an IFN gamma elispot assay. In the publication of 202111, this was used to predict a second reactivation after the first reactivation. Similarly, they used CMV T-track to determine who is at risk for reactivation. In both situations, their assay showed to be successful in identifying high risk patients. In conclusion, monitoring is essential. CMV specific cellular immunity monitoring may allow risk stratification and management of reactivations.

The conclusion of the speakers was:

- Routine, standardized, CMV DNA monitoring by QNAT is essential for aHSCT recipients, especially CMV-seropositive and high-risk patients including those on letermovir, with at least weekly testing in the first 100 days and extended surveillance in higher-risk groups to guide timely pre-emptive therapy
- Standardised CMV-specific cellular immunity monitoring may allow for improved risk stratification and management of recurrent CMV reactivation after HCT.

3. In what ways have newer anti-CMV agents influenced your approach to the early identification and intervention of CMV infection?
The audience was asked how many patients that reactivate cycle through conventional therapies.

This appeared to be more than 60%. The speakers reflected on their local situation. "In 50% of the csCMVI, VGCV is used. In second line foscarnet and others. Especially in countries, like Spain with more limited access, you might not have all options available." says Duarte.

In the Belgian setting, according to Maertens, in the majority of patients VGCV or GCV is used, due to limited availability of alternatives or due to the fear of toxictities. 'The introduction of maribavir changed that.'

Professor Maertens presented a high-level overview of the results of the OTUS EUCAN HCT study, of which the preliminary data was presented at EBMT 2025.[12] This multinational, non-interventional, retrospective, medical chart review (n=79) was set up to gain real-world data on burden of CMV infection and disease in aHSCT patients that developed refractory or resistant CMV infection (R/R) or were intolerant to antiviral therapy (RRI).

Next to R/R patients, this study included intolerant patients as well, which are not in label of maribavir. The results showed a high percentage of nephrotoxicity and myelosupporession both in R/R and intolerant patients (Figure 2).

In the broader OTUS study (including Canada and Israel and other territories as well), they found similar results as in the SOLSTICE data.[13] CMV viremia clearance was associated with reduced rates of myelosuppression (42,6% of patients without vireamia clearance and 17,1% of patients with viremia clearance) and mortality (72,5% of patients without vireamia clearance and 44,8% of patients with viremia clearance).[12]

OTUS EUCAN HCT: Intolerance to anti-CMV therapies post-HCT is driven by myelosuppression and nephrotoxicity

79 adults with R/R CMV or intolerance to anti-CMV therapies (RRI)

10 transplant centres

Follow up 212 months from index CMV episode or until death

	R/R CMVa (n=58)	Intolerance only (n=21)
Nephrotoxicity	32.8%	28.6%
Myelosuppression	62.1%	95.2%

Among patients with intolerance to anti-CMV therapy only following HCT, 95.2% experienced myelosuppression and 28.6% had nephrotoxicity, highlighting a critical and urgent clinical challenge

Figure 2:
Adapted from Bo, T. et al. Abstract A229 EBMT 2025

CMV* cytomegalovirus; HCT, haematopoietic celt transplant; R/R, refractory/resistant; RRI, refractory/resistant CMV infection or intolerance to antiviral therapy.

Maertens L *et at*. Accepted abstract from the 51st Annual Meeting of the EBMT, Florence, Italy, 30 March-2 April 2025 (A228].

The speakers continued reflecting on 2 case studies.

Case study: A 54-year-old male with myelodysplastic syndrome

Recipient
Male
54 years old
First complete remission of MDS-IB2
SORROR score 2
Blood group A-
CMV IgG-positive (CMV PCR negative)

Donor
Male
27 years old
Matched unrelated donor (10/10)
Blood group 0+
CMV IgG-negative

The first patient case (illustrative educational scenario) showed a male patient with myelodysplastic syndrome with a CMV reactivation after discontinuation of letermovir due to positive CMV DNAaemia. Dr. Maertens highlighted to pay attention to patient specific kinetics. The patient received 2 weeks adequate valganciclovir dose, though kept a high viral load. They switched the patient to maribavir, which resulted in a rapid decline of viremia, and no impact on kidney or need for additional growth factor or transfusion. This was a perfect example of maribavir use.

The second patient (illustrative educational scenario) presented a more complicated case.

Case study: A 42-year-old female with acute myeloid leukaemia

Recipient
Female
40 years old
AML in molecular relapse
CMV IgG-positive (Donor/recipient +/+)

The woman, R+D+ with a mismatched donor, was treated for AML. She received 200 days of prophylaxis with letermovir, and after cessation the CMV reactivated at day 234. She was started on foscarnet and antibiotics for multiple opportunistic infections, though developed severe toxicities. She had a low graft function since transplantation, GVHD prophylaxis, prednisolone for acute GVHD, rituximab for EBV reactivation at day 60.

After an increase in creatine kinase and decline in renal function maribavir was started and viremia was cleared within two weeks. Important to note is that they saw no additional toxicities, even with GI GVHD and oral administration of maribavir. Again, in this situation ECIL10 is clear. Maribavir can be started in refractory/resistant infection after 2 weeks of adequate course of an antiviral agent there is no change in viral load (Figure 3).[1]

With that, the presenters shared a brief summary of the pivotal SOLSTICE trial in aHSCT and solid organ transplantation. In all groups, the results showed favor for maribavir vs IAT.[13] These results were recently confirmed in the interim ARISE analysis[14], a real-word evidence study to characterize the effectiveness and safety of maribavir in aHSCT or SOT R/R infections or in patients with intolerance to anti CMV-agents. In this multinational, retrospective cohort study 89 aHSCT patients were included. It showed the CMV clearance rates at the moment of discontinuing maribavir were higher than originally observed in the aHSCT population. Lower incidence of myelosuppression and nephrotoxicity were observed after maribavir initiation, in line with the SOLSTICE trials.[13]

For more information we refer you to poster B073, see page 52.

Both in the EBMT registry, as in RWE studies, there is a growing evidence for use of maribavir in R/R patients, both in safety and efficacy.

The speakers concluded:

- Data from clinical trials have led to the integration of maribavir into current recommendations, influencing treatment algorithms for transplant recipients with CMV infection.
- Phase 3 and real-world studies demonstrated CMV clearance and favourable safety profile with maribavir in transplant recipients, addressing significant unmet needs.
- The availability of maribavir allows for treatment switching in selected patients, with CMV clearance as a treatment goal, consistent with the approved indication

4. What factors influence your clinical decision-making when deciding on an appropriate duration of treatment with maribavir?
According to the label of maribavir, R/R treatment is given for 8 weeks.[15] The presenters concluded that the treatment duration in clinical practice should be based on CMV clearance. The factors influencing the clinical decision-making of an appropriate duration are diverse. Factors like induction regimen, immune monitoring and organ involvement have to be taken into account. Also, the presenters flagged they rather not fully leave a patient without any drug or switch to secondary prophylaxis. It is a very individualized approach.

5. Does the risk of resistance to current antivirals affect your treatment decisions for CMV?
When using maribavir, you have to check for UL97 mutations or UL54 mutations and follow the ECIL guideline.[1] Baseline resistance to maribavir is low, in the SOLSTICE trial it represented approximately 4% of the patients. In those, maribavir is mostly a good option.[13]

When reflecting on the question if resistance impacted the treatment decision, the speakers directly went into the data. They reflected on multiple trials,[16,17] showing higher percentages of treatment-emergent resistance (26%), compared to 4% at baseline in the SOLSTICE. The presenters mentioned that when there is no response under maribavir, it is useful to test for treatment emergent resistance (UL97). Even though the clinical significance of these test remain a question. Clinically it remains hard to differentiate between resistant or refractory. When you suspect resistance: it is recommended genotype, but do not wait for results and treat adequately.

The take home message of the last topic was:
- In patients who develop resistance to maribavir, resistance testing and alternative therapies could be employed to guide CMV management
- Resistance testing for CMV has limited availability and utility, and clinicians should consider proactively switching to maribavir in suspected refractory or resistant cases while awaiting genotypic results

Conclusion
In conclusion, maribavir is beneficial to break cycle of recurrent reactivations:
- Take into account factors as immune monitoring, duration of treatment, use of biomarkers, and potential development of resistance
- It gives a good additional option in our armentarium for these complex patients

Algorithm for management of refractory or resistant CMV infection

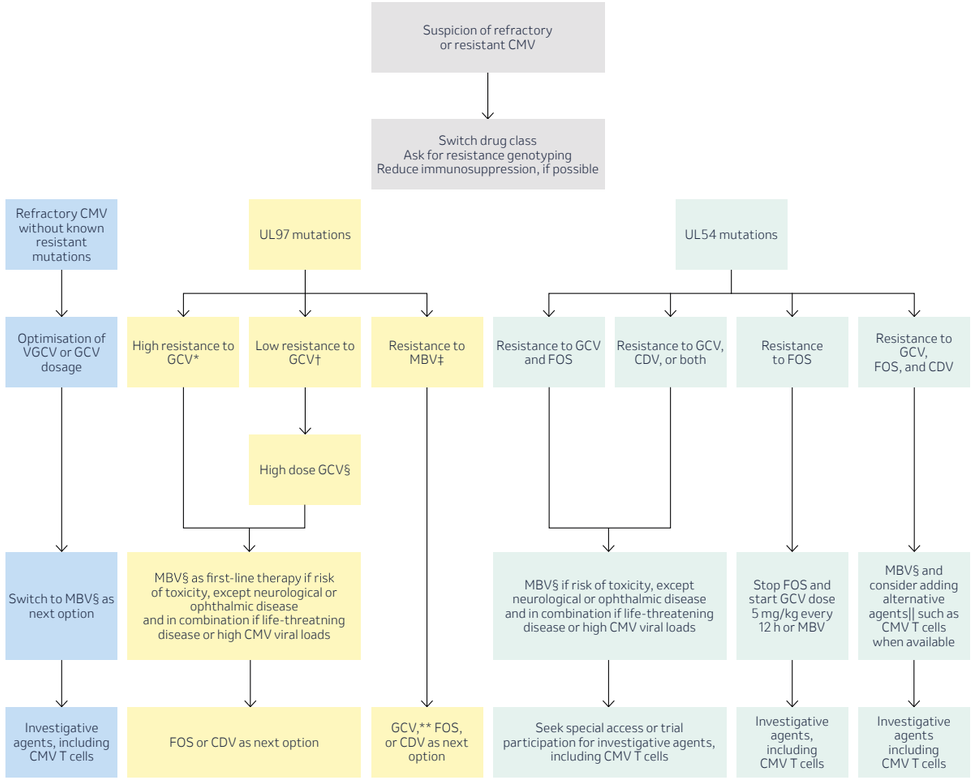

Figure 3: Adapted from Ljungman, P. et al. 2025

Below a reflecton of the discussion following the session	
Would you start foscarnet & maribavir combination in patients with a high viral load to maximize response?	"To maximize response? When starting with a classic polymerase chain inhibitor and then maribavir, it could be less chance to develop resistance with a high viral load. Though data is very limited. Treatment-emergent resistance you see occurring at all timepoints and all viral loads. There is some data to use 1 week a classic antiviral, and then switch to maribavir. You could potentially combine this in patients with CNS involvement. Though in general it is not recommended."
What can you share about stopping maribavir before 8 weeks with quick clearance?	"The medium duration of maribavir use is 7.6 weeks. We know it is common practice to stop earlier, but would only be advised when there is CMV-CMI monitoring. Viral load clearance is an indicator for survival."
What to do when resistance occurs under maribavir use?	"Donor lymphocyte infusion could help for specific immunity. Also, switching to valganciclovir or foscarnet depending on individual patient characteristics of course."
How long to keep monitoring after ceasing prophylaxis? Should you continue until immune reconstitution is in place?	"There is not one exact assay for immune recovery in place. After 100 days prophylaxis, it would be common practice to monitor bi-weekly. It also depends on corticosteroid use, cellular immunity and lymphocyte count."
What is the role of allo CTL products?	"When there is no CMV-CMI and the patient reactivates again, third donor T cell adoptive T cells could be considered. The guideline states in case of very severe CMV. But you could also consider this in immunosuppresed patients where you want to boost the immune response."
How about the development of CMV vaccines?	"Some candidate vaccines look promising in phase 3 trials. In siblings it seems doable, in unmatched patients it is harder. You should consider who to vaccinate, the donor or recipient? It is hard to design. Currently, there is also development of new mono & polyclonal antibodies cocktails. Things are moving."
Since most of us do not have assays in place, what markers could we use for CMV-CMI?	"You could look at immune response. When CMV specific monitoring is not available, you can look at immunoglobulins or CD4. It is more aspecific though, so it's an indication of severity and should not be solely used for a treatment decision."

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MSD Industry Symposium IS16
March 22nd, 2026

Optimizing CMV Management in Pediatric and Adult HSCT: Recent Evidence and Clinical Implications

The burden of CMV in transplantation: Past lessons and major milestones

Rafael de la Cámara

Dr. de la Cámara reviewed the evolving burden of cytomegalovirus (CMV) in transplantation, opening with the provocative question of whether CMV risk is increasing or decreasing. Interesting enough, the audience answered very similar for both an increase or decrease. And although the majority of the attendees answered that all aHSCT patients are at high risk for CMV and candidates for letermovir prophylaxis, there was also a group that recognized a subgroup of patients at low risk of CMV. Dr. de la Cámara emphasized that the severity of CMV disease still must not be underestimated, and all CMV-seropositive aHSCT patients should be considered for prophylaxis.

"We should never forget the severity of CMV infections", de la Cámara says. He consequently provided a nice overview[1-5] of the evolution of CMV management with historical perspective and milestones:

- 1st era: health care professionals underestimated CMV's clinical impact
- Dark era: 20-30% non-relapse mortality was caused by CMV
- Pre-emptive era: because of the development of diagnostic methods and CMV-antivirals for pre-emptive therapy, attributable mortality has fallen to ~2-3%.

"So, was the problem of CMV been solved then?" Dr. de la Cámara asked rhetorically. Available drugs are associated with myelosuppression, delayed immune reconstitution, and/or nephrotoxicity, and are not suitable for prophylaxis.[6-10] "Nowadays more patients die due to the indirect effects of CMV then due to CMV disease."

The fourth era, from 2018 onwards, is marked by the introduction of letermovir as CMV prophylaxis, abrogating the mortality gap based on CMV serostatus[11] (Figure 4).

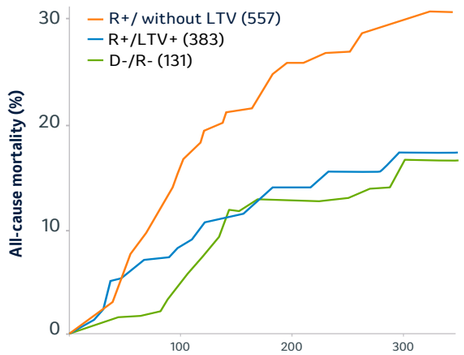

Figure 4: Adapted from Febres-Aldana, A. et al. (2024)

Are the risk factors for CMV infection/disease in aHSCT increasing?
The short answer: yes. The main changes seen in the last years resulting in an increase of CMV risk are the increasing age of patients, more haplo and unrelated donors, and the increased use of PTCy for GVHD prevention, where PTCy has shown to double the risk. But also the increase in the number of CMV R+ recipients and mismatched donors are a factor, as well as the increased combination of R+/D-.[12,13,14]

Milestones
When reflecting on the milestones in CMV management, de la Cámara highlighted that letermovir, next to aHSCT, now has been approved for kidney transplant and pediatrics as well.
He showed a table with publications on 200 days duration of prophylaxis, post hoc analysis mortality, RWE and in pediatric patients.
Also, maribavir for R/R infection helped mitigating the CMV risk.[15-22]

He gave a short summary of the phase 3, randomized, double-blind study of Marty et al[15] in 570 patients. The primary endpoint was the proportion of patients, among patients without detectable CMV DNA at randomization, who had clinically significant CMV infection through week 24 after transplantation. This study did not only demonstrated less viremia, but also significantly later (40 days placebo vs 139 days, p=0.0173).
The Green paper[23] was a retrospective, single-center cohort study in 926 aHSCT patients who were CMV seropositive or had a CMV seropositive donor, investigated the association between cytomegalovirus viral load and mortality in the first year after haemopoietic stem cell transplantation. The authors concluded that any level of CMV-positive PCR can increase the level of mortality, but also that early reactivations have a higher impact: >250IU/mL (1.000 copies) increased mortality by 20-fold in the first 60 days vs <2 fold later. "So", concluded de la Cámara, "it is an advantage to have late versus early CMV replication."

Maribavir gives an additional option in the armentarium to treat R/R CMV infection, as was seen in the SOLSTICE trial.[18] This phase 3, open-label study in 352 aHSCT and SOT recipients with R/R cytomegalovirus investigated confirmed cytomegalovirus clearance at end of week 8. Maribavir showed more effective, with lower toxicity compared to foscarnet and galvanciclovir/valganciclovir. But a proportion of patients still fail or reactivate, requiring further treatment. One of the potential explanations is treatment emergent resistance (4/234: 1,7% at baseline, and 60/234: 26% after it's use). This was associated with lower response (49/103: 48%).[18,24]

Can the broad implementation of letermovir as prophylaxis have an impact on other infections, for example on HHV-6 or bacterial infections? Two retrospective studies[25,26] showed that this is not the case for HHV-6. A retrospective study from Japan[27] aimed to examine the relationship between letermovir prophylaxis and the incidence of bacteremia and invasive fungal infections, using a Japanese transplant registry database.
They analyzed 19,531 patients (4915 patients in the letermovir group and 14,616 in the non-letermovir group) who underwent their first allogeneic HCT from 2011 to 2022. The results showed a significant decrease bacteremia at day +100 of 17,4% vs 21,7% (p<0,001, HR 0,75, CI 0,69-0,81) in the letermovir group versus the non-letermovir group.

In summary, the risk of CMV is increasing, and preventing CMV damage is important. Prophylaxis is widely recommended to a minimum of 100 days, as per ECIL 10, but also the US and Spanish guideline. Maribavir is an advancement in the treatment of R/R CMV. Still some challenges remain, which are discussed by the next two speakers.

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Evolving strategies and remaining challenges in CMV management in adult HSCT

Per Ljungman

Before discussing the remaining challenges, Prof Ljungman asked the audience when they start prophylaxis. This is straight forward, as early as possible. More variation is seen in the duration in high risk patients; the majority of countries can give only 100 days, but 30% of respondents indicated that prophylaxis is given for 180-200 days post-transplant.

Testing for CMV-specific cellular immunity is standard practice for 27% of the audience, but 46% do not have access to these tests.

When to start
Ljungman then discussed the first challenge; the uncertainty when to start prophylaxis. "To be fair", says Ljungman, "no prospective studies analyzing long term results comparing time to start prophylaxis." Ljungman shares his personal interpretation of the existing data: the biggest advantage of starting early is to not miss early CMV, and the lower rate of 'blips'. On the other hand this comes with disadvantages associated with costs and potential compliance due to nausea directly after transplant. In the pivotal study[1] 18% of the patients screened could not be included in the trial due to early CMV, and almost 10% (n=70) were CMV PCR positive at study entry. The study showed that patients with detectable CMV DNA had a higher rates of csCMVi in both arms. "Early reactivation is not uncommon", Ljungman concluded, also highlighting the conclusion of Green et al.[3] that any level of viremia is associated with mortality.

Due to variability in monitoring techniques and patient cohorts, and 'blips' it is not possible to set a fixed threshold when to start PET.[2] In this respect it is also important to separate 'real' reactivations from 'letermovir blips' (note: blips also do occur in the absence of letermovir).

"In the publication of Einsele et al.[4], it is extensively described how to treat blips." Ljungman continued. "When blips are measured, there is some viral DNA synthesis, but no production of full virions. One of the most recent innovations is a new assay, developed by an Italian group, using UL-21.5 mRNAemia to distinct between blips and reactivation."[5,6]
Ljungman presented an interesting table how he distinguishes 'real' reactivations from 'blips' in case of letermovir use:

	'Real'	'Blips'
Time after start of letermovir	Frequently late	Early after start of letermovir
Increasing viral load	Common	Less frequent
'High' viral load	Common	Rare/absent
Number of positive tests	Several	1 or max 2

Duration
The second challenge that was raised, is the duration of prophylaxis. In the pivotal trial, the duration of prophylaxis was 100 days. The data showed that reactivations occur after discontinuation of letermovir. The subsequent randomized, double-blind, placebo-controlled trial from Russo et al[7], compared no extended prophylaxis to 200 days prophylaxis (n=220). A secondary endpoint was the time to develop csCMVi until week 48. As was expected, when ceasing letermovir no difference in occurrence of CMV reactivation was seen, but the time to csCMVi differed significantly in favor of prophylaxis (p=0.0001).[7]

Concluding on the length of prophylaxis, most of the patients don't need extended prophylaxis.
Those patients who are clearly high risk, "the trouble makers" as Ljungman says, or when you see poor immune reconstitution, 200 days can be beneficial.
"It remains an individual risk assessment, and more studies into CMV-CMI are necessary." Ljungman concluded.

EBV
The next hot topic is EBV. The publication of Camargo et al[8] includes a nice summary of available data.
Prof. Ljungman noticed 7 from the 8 studies are from China. The paper of Shafat et al[9] showed no significant correlation between EBV and PTLD (post-transplant lymphoprolipherative disorder). It is clear that ATG is a risk factor for EBV infection.
The high use of ATG in China and a cultural disposition of Asian ethnicity might explain the geographical differences. Ljungman concluded that in most circumstances the advantages of letermovir prophylaxis by far outweigh the EBV risk.

Implementation of CMI testing
The last 'challenge' covered by Prof. Ljungman was CMV specific immune response. The use of antiviral agents delay CMV specific cell mediated immunity, because of the lack of antigen exposure. Ljungman referred to an older study from Chemaly et al[10], showing that limited CMV-CMI resulted in a lower survival probability. Though there is no clear consensus, due to the lack of studies with an D+/R- comparator, varying csCMVi definitions, variability in tests and timeframe of testing, and the potential impact of transplant-specific risk factors, the ECIL-10 guideline committee[2] include the optional use of a CMV-CMI assay. Currently, a Quantiferon assay could not effectively predict csCMVi development in patients who received letermovir prophylaxis.[11]
Later this congress data from China will be presented from an RCT to predict csCMVi risk after stopping letermovir with the help of CMV-CMI, see oral session of Wang et al, p28.

Suppression of CMV does not increase the risk for late CMV disease, despite lower CMV-specific immunity, as was seen by Sadowska-Klaska[12].
Late CMV disease occurred most frequently in a setting of prolonged low-level untreated viremia and was independently associated with death 2 years after transplant. However, these results do no automatically mean all low-lever reactivations need antiviral treatment. The existing data whether immune monitoring can help predict which patients need treatment is still insufficient [personal interpretation of data].

In his personal opinion, professor Ljungman proceeds that CMV management remains a long-term strategy. A patient should be successfully managed until immune reconstitution occurs without developing CVM end-organ disease, preferably without any reactivation. CMV disease is a failure of the CMV strategy.

"For now CMV-CMI would not be sufficient to guide treatment, but it could be a long term strategy for adequate use. We need well designed, prospective, interventional trials with relevant clinical endpoints to further interpret CMV-CMI testing in practice. And of course, it will be interesting to see how the vaccines trials will develop." concluded Ljungman.

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From evidence to practice: Advancing CMV management in pediatric HSCT

Dina Averbuch

The last speaker, Dina Averbuch from Israel unfortunately could not come to Madrid, though pre-recorded her session, sharing her experience with CMV in pediatric patients.

Although results of studies vary, approximately half of the pediatric patients experience a CMV re-activation after aHSCT[1], and an average of ~4,5% (range 0-12.9%) develop CMV disease.[2-10] This is associated with poor graft function, chronic GVHD and higher NRM.[11-15] The biggest risk factors for CMV infection and disease in children were demographics as older age and male sex.

But also seropositivity, malignant underlying disease, unrelated/mismatched, haploidentical or cord blood donors, the use of ATG or alemtuzumab and grade III-IV GVHD and delayed CMV-specific cell-mediated immunity (CMI).[1,2,6,7,16-27]

PET in children
The advise in ECIL-1029 is to use IV ganciclovir or foscarnet or oral valganciclovir as first line PET. Dr. Averbuch first reflected on two studies about the initiation of PET in children.

1. Camacho-Bydume[16], a retrospective, single-center study from 2022 in 169 children, analysing the impact of the timing of induction treatment on overall survival and CMV disease, concluded that the timing of induction treatment was significantly associated with overall survival (p=0,02), and that the optimal time to start PET is within a week after CMV infection
2. The recent prospective interventional study from the UK by Duret et al, 202530 aimed to determine an optimal viral load threshold for initiating PET in paediatric HSCT patients and test the impact of this threshold in real- world practice. CMV disease and mortality among 215 aHSCT pediatric patients (50% with inborn errors of immunity) was reduced from respectively 23,1% to 4,1% and from 30,8% to 19,4% when the viral load threshold for initiating PET was reduced from 2500 IU/mL to 1000 IU/mL in pediatric patients.

Averbuch then presented a case study of a young girl, a tough case:
The 5-year old, transplanted after a relapse B-ALL, experienced a CMV-reactivation at day 20 (before engraftment). In line with the ECIL-10 guideline foscarnet was started. Unfortunately she appeared to be foscarnet intolerant, with severe side effects (among which several abdominal pain, vomiting, urethritis, and acute kidney injury). After switching to ganciclovir, she developed neutropenia and a life threatening sepsis. What alternative options were available in this situation? Maribavir was not easily available in our country, and there was no access to adoptive physical therapy. Immune suppression could not be reduced, because she developed GVHD in her skin and she also had gastrointestinal implications. An endoscopy showed severe inflammation of mucosa and ulcers and purulent discharge. The biopsy and PCR were positive for CMV, and maribavir was started.

In the end, this child was exposed to four lines of CMV treatment, severe toxicity of preemptive therapy, and she still developed CMV disease. This raised the question whether prophylaxis could be indicated in these situations.

Prophylaxis in children
What are the considerations when using prophylaxis in pediatric patients? For example, is letermovir efficient and does it have a manageable safety profile in children? Or when to start and what dosis? The prospective phase 2B open label study that assessed pharmacokinetics, safety and efficacy of letermovir in 63 children from 40 centres across 11 countries showed similar levels of exposure to thereapeutic dosages of letermovir compared to adults, with or without cyclosporin. With the exception of three children in the youngest group who had lower levels. Following this observation, the dosages in younger children was increased.[31]

8,2% developed csCMVi during treatment with letermovir and another 4,3% after discontinuation of prophylaxis. No cases of CMV disease were seen. The tolerability was manageable with few gastrointestinal side effects (vomiting 17.5%, and nausea 3.2%). Two children (3,2%) discontinued letermovir because of they developed serious drug-related side effects (atrial fibrillation and increased blood bilirubin, both multifactorial and resolved). Two patients developed UN56 mutation, one of them started letermovir with detectable viral load.[31] Based on this study daily dosage of letermovir was recommended by weight bands (5kg to <7.5kg, 7.5 kg to <15 kg, 15kg to <30kg, and ≥30kg).[32]

What should we know before deciding on prophylaxis in children

- **Real world efficacy**
  Data on prophylaxis in pediatric patients remains limited. Approximately 20 retrospective studies exist, of which 17 single centre studies.[1,3-6,9,33-46] In total, these studies included 664 children. The majority of the studies showed a significant difference between letermovir and historical control groups on CMV reactivation (12% [0-27,8%] vs 50% [29,5-77,4%], 6 studies) and on csCMVi (10,7% [6,6-12%] vs 44,9% (17-64,5%], 5 studies). “The studies showed that letermovir has similar efficacy results in pediatric patients as in adults.” says Averbuch.

There were no cases of CMV disease among almost 500 children treated with letermovir (reported in 15 of the studies), there was no significant difference with the control groups. Transient CMV DNAemia during prophylaxis occurred in 11,1-22,7%. ”But this did not lead to the interruption of prophylaxis.” Averbuch added.

- **Toxicity**
  The rates of toxicity were low. The main adverse effects that were seen were nausea and vomiting, and rarely liver enzymes elevation.

- **Serostatus**
  Both the European ECIL-10 guideline[29] as the American ASTCT guideline[47] recommend primary prophylaxis with letermovir as the strategy of choice for primary prophylaxis against CMV in CMV seropositive children following allogeneic transplantation. ECIL-1029 clearly states that prophylaxis in patients with CMV seronegative status is not recommended.

- **Start**
  According to the recommendations[29], prophylaxis should be started as early as feasible, but no later than 28 days after transplantation. There was not enough data to rate this recommendation. "Usually, prophylaxis is started within one month after transplant, when children are able to swallow, intravenously or when oral granules are available. Though in pediatric studies there is a big range when prophylaxis is started." said Averbuch. A recent Chinese study33 showed that later initiation of letermovir administration was a risk factor for the occurrence of csCMVi (HR 1,07, 95% CI 1,01-1,14, p=0,029).

- **Duration**
  In guidelines[29,47], the general recommendation is to continue prophylaxis for 100 days with the possibility of extended prophylaxis for children who remain at risk. After discontinuation of letermovir 0-12,8% of children develop csCMVi.[4,38] Studies show a large variety in the duration of prophylaxis, including duration based on immune recovery.

- **Influence on immune reconstitution**
  Immune reconstitution in children who received letermovir should be further investigated. There is some data in adults demonstrating that prophylaxis delayed immune reconstitution. Dr. Averbuch presented a Chinese publication with 80 children who received prophylaxis with letermovir and compared this to 98 controls. On day 90 post-transplant the letermovir group showed significantly lower levels of lymphocytes (p<0,001), T cells (p<0,001), CD4 - (p=0,002) and CD8 cells (p<0,001).[34] However, in a multicenter study performed in Italy, matching 80 children on letermovir to 80 controls on key covariates influencing immune reconstitution (age, donor type, ATG, aGVHD, pre-HSCT CMV serostatus) no difference in the lymphocyte subsets was found.[4]

To summarise, clinically significant CMV has a major influence on the course of aHSCT in children. Available first line treatment options are associated with toxicity. Letermovir decreases the rate of csCMVi, with limited toxicity.

But there are still several knowledge gaps. There is limited data on experience in children less than two years of age. There also is a need for more information on potential resistance due to potentially lower exposure, as well as on immune reconstitution. And another important aspect is the specific management of children with primary immune deficiencies.

More, specific, pediatric data are essential to further establish optimal and safe treatment strategies.

a. The speaker noted that some children with inborn errors of immunity may be CMV-seronegative yet infected; this is off label, and this observation is noted for awareness only and is not discussed further or intended as clinical guidance.

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Below a reflection of the discussion	
What is the role of therapeutic drug monitoring in the management of CMV?	"TDM is common practice for cyclosporin, vancomycin, azoles and more. With VGCV it is not common practice. However, it is easy to underdose in case of VGCV. This risk is even larger in pediatric patients. So, if TDM is available in your center, use it."
How long to give maribavir in refractory/resistant CMV infection?	"Maribavir should be given 8 weeks according to the label. During treatment you should check for resistance and change the drug if necessary."
Do you routinely check for GCV resistance?	"Follow the ECIL guideline, for all refractory infections, it is valuable to send it for resistance testing. Especially for down the line decisions, the results are mostly not used for ad hoc decisions."
Currently in refractory infection we mostly use old school strategies. What else could I consider?	"There are not enough studies. There are currently some new initiatives, for example T-cell infusions for refractory infections. Since there are less cases of CMV disease due to prophylaxis, trials become harder."
What is your recommendation in GVHD and immunosuppression?	"The oral component can be limiting for some patients with gastro-intestinal complaints. It is still unknown if CMV is driving GVHD or the other way around."
How do you reflect on long term prophylaxis?	"When using letermovir for a duration of 180-200 days, there was no increase in toxicity. It is difficult to recommend anything, without sufficient data to support. Personally, there is no reason not to prolong prophylaxis."
Is there a gender difference?	"No"
What is the role of determining serology before conditioning and defining therapy?	"Serology is normally tested. There are multiple studies, from Geneva, the UK, and Toronto, that show an overestimation of seropositivity when measured closer to transplant compared to moment of diagnosis (20-25%). Also, there are 2-3% of false negatives. There is a difference in pediatric patients too. In the most recent update of the ECIL guidelines, the word serostatus has been changed to CMV status. To prevent these event."
How do you see use of lintuzimab before transplantation?	"There is only limited data on new drugs. There are 2 available ECIL papers on lintizumab, though not looking specifically into CMV after transplant and the role of immune reconstitution. This also triggers the question on potential vaccines. Thought the design of this studies are not easy, to trigger an immune response. Currently there is a liver transplant study with a vaccine ongoing in the US."

Scientific Sessions

Pharm1 - Pharmacists Day 1 | Clinical update

Chairs: *Nick Duncan (Birmingham, United Kingdom of Great Britain and Northern Ireland (the)), Estela Moreno Martinez (Barcelona, Spain)*

Beyond CMV - management of viral infections in the transplant setting

Speaker: *Rafael de la Cámara (Madrid, Spain)*

Dr. de la Cámara started his presentation by emphasizing the importance of having specific knowledge of infections in the HSCT setting, as extrapolation from other patient populations is not appropriate. He highlighted that viral infections in the transplant setting follows phase-associated patterns, with certain viruses such as HHV-6, EBV, and VZV showing 'exclusive' presentations in this context. In addition, he stressed the need to use updated, disease-specific definitions, referring to IFI (2020)[1], CMV (2024)[2], and HHV-6 (2019)[3]. Overall, viral infections represent the most frequent infectious complications after HCT.

He continued by addressing the challenges in diagnosing infections in this population. A proven diagnosis often requires biopsy, which is not always feasible. Moreover, understanding the interpretation of diagnostic tests is essential. Importantly, he underlined the importance of distinguishing infection from disease: while infection is diagnosed in the laboratory, disease represents a clinical diagnosis requiring integration of multiple parameters.

Dr. de la Cámara then discussed the appropriate use of management strategies. He outlined three main approaches: prophylaxis, empirical therapy, and pre-emptive therapy, in addition to targeted treatment when a pathogen is identified.

He subsequently focused on the importance of prevention of infectious complications for achieving good outcomes after aHSCT. Two major advances have contributed to improvements in this field. First, better GVHD prevention, evolving from ciclosporine and HLA typing in the past to the current use of PTCy. Second important change in supportive care is the development of pre-emptive therapy for CMV, and now prophylaxis with letermovir. These developments are also reflected in clinical outcomes. "CMV-related mortality has declined substantially over time", said Dr. de la Cámara, "CMV control is probably the single advance with the highest impact on transplant survival over the past 30 years."[4-7]

He further illustrated that infections after SCT follow a phase-associated pattern. Historically, CMV and P.jiroveci were typically observed in the post-engraftment phase (phase II). "These have now shifted to the late phase (phase III)." according to De la Cámara.

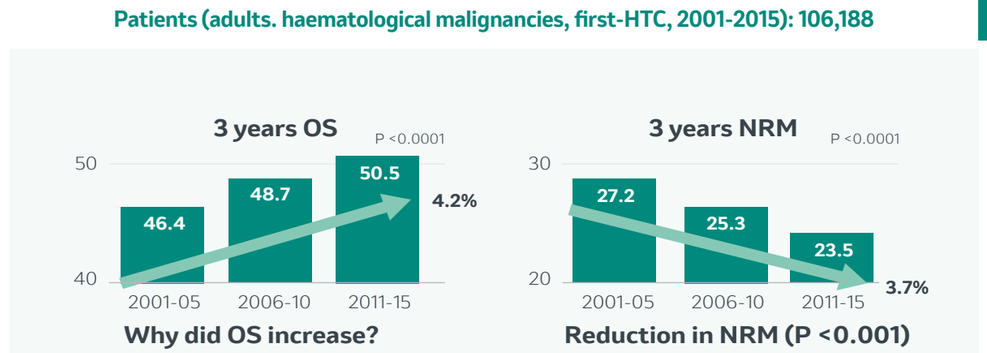

Figure 5: Adapted from Shouval, et al. 2019

Scientific Sessions

Data from an international registry (CIBMTR, 2019–2023) further highlights that, both in allogeneic and autologous transplants, infections remain the second most important cause of death after aHSCT, both within 100 days and beyond 100 days post-transplant. These data included only deaths due to infection in the absence relaps, active GVHD or GVHD treatment.[8] He raised the question, however, what the contribution of infections is in patients who relapse or have GVHD.

To emphasize this, data from a large cohort of 106,188 adult patients with haematological malignancies undergoing first HCT (2001–2015) was shown. In this cohort an increase in overall survival of 4.2%, from 46.3% in 2001 to 50.5% in 2011 ($p<0.0001$) was found. This was accompanied by a reduction in non-relapse mortality of 3.7%, from 27.2% to 23.5% ($p<0.0001$) (Figure 5). No decrease in relapse was observed over time, except in cord blood transplantation (30.8%, 34.7%, and 28.7%, respectively).[9]

Dr. de la Cámara referred to ECIL as a common initiative of EBMT, EORTC, European LeukemiaNet and ICHS, aimed at developing European guidelines for the diagnosis, prophylaxis, and treatment of infectious complications in leukemic patients. In that context, he concluded that management of viral infections in the HCT setting can be viewed as a structured approach, in line with ECIL principles, combining preventive and therapeutic strategies. Prevention includes donor selection based on serology (CMV, EBV), personal hygiene and protection against community-acquired viruses (including CMV and norovirus), vaccination (influenza, RSV, MMR, HPV, polio, HBV, and others such as TBE and yellow fever), antiviral prophylaxis (HS, HZ, CMV, HBV, and EBV with rituximab), and PET (CMV, EBV, HBV). Treatment is required in case of failure of prevention (e.g. CMV, EBV, HS, HZ, influenza, RSV) or in infections for which no satisfactory preventive strategies are available, such as HHV-6, BK virus, and JC virus.[3,10,11]

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IDWP - Infectious Diseases Working Party Session
March 23rd

Chairs: *Dina Averbuch (Jerusalem, Israel) – Rafael de la Cámara (Madrid, Spain), Jan Styczynski (Bydgoszcz, Poland)*

Refractory / resistant HSV and CMV – an underestimated problem?

Speaker: *Per Ljungman (Stockholm, Sweden)*

Resistant and refractory infections are not the same. “An important distinction that is often misunderstood in clinical practice.” said Ljungman. We see difficult to manage patients where we cannot deliver the proper antiviral dosing due to toxicity or concomitant medications, but this should not be classified as refractory.

He stressed that viral infections are often too quickly labelled as refractory. In this context, he explained the rationale behind the commonly used 14-day timeframe for defining refractory CMV infection. He explained that antiviral therapy requires time to first slow and subsequently stop viral replication. As an example Ljungman refers to a RCT by Mattes et al.[3], comparing intravenous ganciclovir with oral valganciclovir, he showed that viral load decline typically becomes apparent only around day 17–20, with little effect observed before day 14 (Figure 6). He cautioned that changing therapy too early, particularly to a drug with a different mechanism of action, may reset the antiviral effect and potentially be harmful.

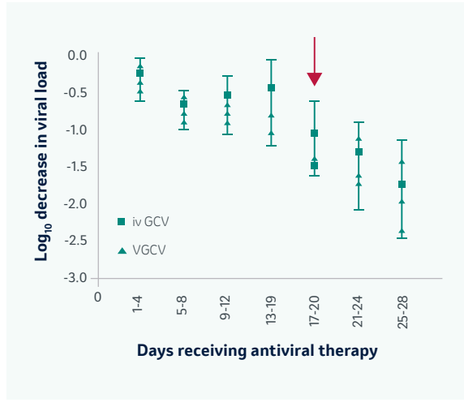
Prof. Ljungman further discussed antiviral resistance mechanisms. Different anti-CMV drugs have different thresholds for resistance, related to their mechanism of action. Previous antiviral exposure increases the risk of resistance, although resistance mutations can also occur in naïve CMV strains. In addition, although rare, multiple sequence polymorphisms of uncertain significance exist, but mutations in relevant genes may confer varying levels of resistance. Together, these factors increase the risk of underdosing, should you not be aware of this. The main CMV genes involved in antiviral resistance, including UL97, UL54, UL27 and UL51/UL56/UL89.4

Data from clinical studies were presented to illustrate the frequency of antiviral resistance. In the AURORA study, comparing valganciclovir and maribavir for primary PET, resistance mutations at baseline were rare (2/547 patients with valganciclovir resistance). After treatment, resistance increased to 2.5% in valganciclovir -treated patients and 10% in maribavir-treated patients.[5]

In the SOLSTICE study, including SOT and HSCT recipients with resistant/refractory CMV infection, baseline resistance was already present in a substantial proportion of patients. UL54 mutations conferring ganciclovir resistance were observed in 34/304 patients (11.2%), and foscarnet resistance in 18/304 (5.9%). UL97 mutations associated with ganciclovir resistance were present in 179/310 patients (57.7%), while maribavir resistance mutations were rare (3/310; 1%). After investigator-assigned therapy, 16/106 patients (15%) developed resistance mutations, compared with 60/234 (26%)

	CMV	HSV
Resistant	A resistant CMV infection is defined as a refractory infection in combination with viral genetic alterations that decreases susceptibility to one or more antiviral drugs.[1]	A resistant HSV infection requires a refractory clinical course together with viral genetic alterations and/or phenotypic evidence (e.g. EC50 above assay cut-offs) indicating reduced susceptibility to antiviral therapy.[2]
Refractory	CMV viremia (DNAemia or antigenemia) that increases (ie, >1 log10 increase in CMV DNA levels in the same blood compartment from the peak viral load as measured in the same laboratory and/or with the same commercial assay) or persists (≤1 log10 increase or decrease in CMV DNA levels) **after at least 2 weeks of appropriate antiviral therapy**	The lack of clinical improvement in HSV-positive mucocutaneous lesion(s) **after at least 7 days of appropriately dosed, directed anti-HSV therapy**, in the absence of other plausible causes of mucositis, such as recent high dose chemotherapy, irradiation, oral GVHD, fungal or other viral infection – or – the occurrence of a new HSV-positive mucocutaneous lesion(s) after receiving appropriately dosed, directed anti-HSV therapy **for at least 7 days** (excluding prophylaxis and suppressive antiviral therapy)

Figure 6: Adapted from Mattes et al. 2005



after maribavir treatment (17% in responders, 46% in non-responders, and 86% in patients with response followed by rebound).
This showed that with more advanced disease, more advanced therapy they have received, the higher the risk of resistance.[6] However he noted that with the introduction of letermovir, resistant/refractory CMV is now observed less frequent than before the letermovir era.[7]

Across multiple studies, letermovir resistance appears to be uncommon in patients receiving prophylaxis.[8-12] We do not know what the frequency of letermovir resistant-infections would be when used as treatment, since this is not recommended. The likelihood of driving resistance differs between antiviral agents. “Maribavir is relatively more likely to drive resistance than ganciclovir/valganciclovir, whereas foscarnet, cidofovir and brincidovofir are less likely to do so.” Ljungman said. “No comparison can be made with letermovir, because it is not fair to compare PET with prophylaxis. Also, this needs to be balanced against toxicity and the overall clinical context.” The ECIL-10 guidelines include an algorithm with available options in case of multidrug resistance.[13] An effective option for multiresistant patients is virus-specific T-cell therapy, with reported response rates of approximately 70% after HCT, although with a wide range (30–100%).[14]

The main genes associated with HSV resistance include UL23, UL30, UL5 and UL52. Resistance is relatively uncommon, occurring in 0–1% of immunocompetent patients and 0–14% of HSCT recipients. Identified risk factors include recurrent infections, high-risk transplants (haploidentical or cord blood), HSV-seronegative donors, prior HCT, HSV-2 infection, and, most important, inadequate or intermittent antiviral therapy.[15]

Clinically, resistant/refractory HSV most often presents as mucocutaneous disease associated with pain and reduced quality of life. More severe manifestations, such as visceral disease, are rare but have been reported. Resistant/refractory HSV has been associated with poor outcomes, including renal injury, hospitalization, and death.[15-18]

Ljungman: “Management strategies depend on the level of resistance. In high-level acyclovir resistance (UL23 mutations), foscarnet or cidofovir can be used. In low-level acyclovir resistance (UL23 mutation), dose escalation or continuous infusion of acyclovir, or use of foscarnet/cidofovir, may be considered. In cases of multidrug resistance (UL30 mutations), virus-specific T-cell therapy or pritelivir may be options.” However, evidence for VST is mainly limited to case reports, and pritelivir is still under development (phase 3 results were presented at the conference in another session).[19]

Prof. Ljungman concluded that antiviral resistance is not uncommon and may lead to prolonged infections and adverse outcomes after aHSCT. However, not all refractory infections are resistant, and resistance testing is therefore an important tool in the management of patients who are perceived to be refractory to antiviral therapy.

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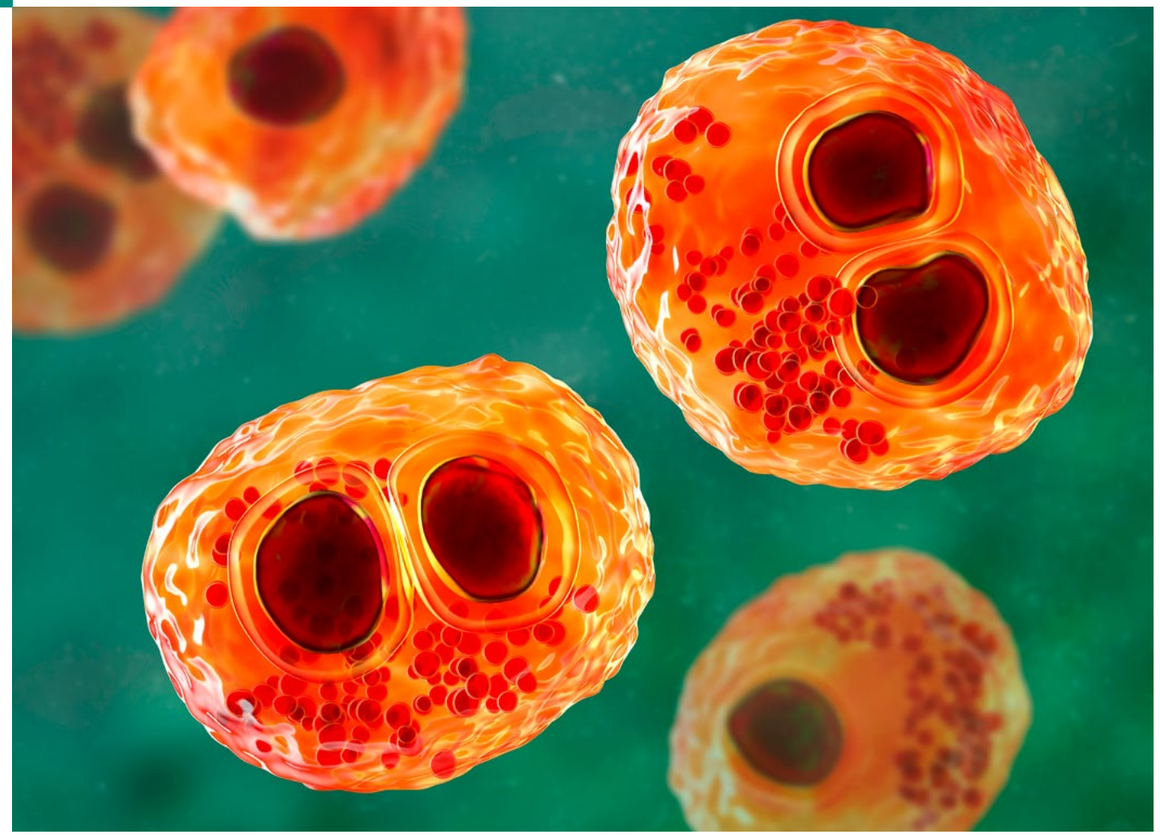
Specifics of infections management in patients following post-transplantation cyclophosphamide

Speaker: *Montserrat Rovira (Barcelona, Spain)*

The use of post-transplant cyclophosphamide (PTCy)-based GVHD prophylaxis has expanded beyond its original use in haploidentical donor transplantation. Dr. Rovira showed that PTCy is increasingly being applied in mismatched unrelated donor (MMUD) and matched related donor (MRD) transplant settings, both in the United States and in Europe.[1] In this context, she referred to the 2024 EBMT consensus recommendations, which aim to standardize prophylaxis and treatment strategies in aHSCT, including the use of PTCy.[2] "For example, in our own institution, Hospital Clínic Barcelona", said Rovira, "over the past two decades the use of PTCy increased from 0% in 2011 to approximately 90% in 2023, across different type of transplants."

In a matched-pair analysis of 200 patients comparing CsA-based versus PTCy-based prophylaxis in MSD peripheral blood aHSCT (2017–2021), Dr. Rovira showed that PTCy is associated with delayed hematopoietic recovery. Neutrophil engraftment occurred later (median 16 vs 13 days), and time to transfusion independence was prolonged (RBC 22 vs 12 days; platelet 20 vs 11 days). [3] In addition, delayed immune reconstitution was observed.[4] This delayed immune recovery is associated with a higher incidence of infectious complications in patients receiving PTCy-based prophylaxis (Figure 7).[5]

In particular, CMV reactivation has been reported more frequent in this setting. "But", according to Rovira, "with the use of letermovir prophylaxis this risk is manageable in current clinical practice."[6] This was illustrated with a study by Brusosa et al., where a clear downward trend in CMV-related events was observed following the introduction of letermovir prophylaxis, despite the increased infectious risk associated with PTCy-based transplant strategies.[7] Beyond CMV, other infectious complications were also observed more frequently.



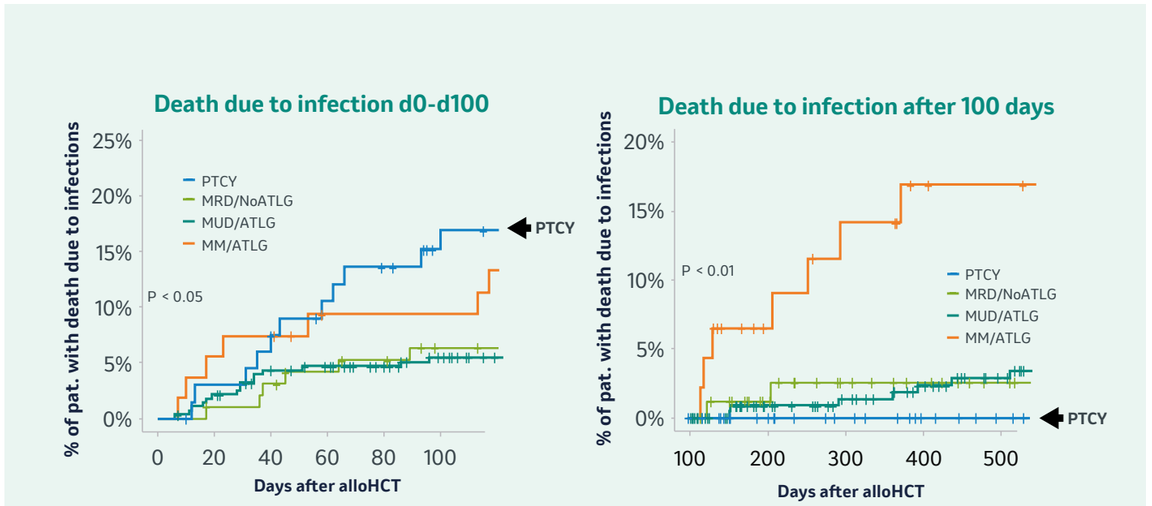


Figure 7: Adapted from Meyer et al. 2025

An increased incidence of BK virus-associated hemorrhagic cystitis was also noted, particularly in the haploidentical setting. However, most cases were manageable with supportive care and rarely progressed to severe (grade 3–4) toxicity.[8]

Fungal infections appeared to have a relatively low incidence in the context of PTCy, both for yeasts and molds, although invasive mold infections remained associated with higher mortality.[9] Bacterial infections commonly occurred in the early post-transplant phase: in the presented analysis, 37% of patients developed a bacterial infection, of which 70% within the first 30 days, involving both gram-positive and gram-negative pathogens.[10]

Optimization of the stem cell product can mitigate infectious risk; in the haploidentical setting, infusion of >5 × 10^6/kg CD34+ cells has been associated with improved overall survival.[11] Alternatively, the use of G-CSF starting from day 7 was shown to reduce bacterial infection rates without an apparent increase in endothelial complications, and with comparable incidences of GVHD, SOS, TA-TMA, and survival outcomes.[12] Dr. Rovira also stressed the importance of dedicated supportive care, including central venous catheter management, management of gastrointestinal toxicity, and rectal screening.

In conclusion, PTCy can be an attractive and broadly applicable strategy for GVHD prophylaxis irrespective of donor type. At the same time, optimization of supportive care and infection prevention remains critical, as well as accelerating hematopoietic recovery.

During the discussion, the question was raised whether the dose of PTCy or combination with other drugs influences the infection risk. Dr. Rovira noted that the presented studies were conducted using a dose of 50 mg/kg cyclophosphamide, but suggested that dose reduction in selected settings may shorten the duration of neutropenia.

Further research is needed to determine in which settings dose adjustments and combination strategies may reduce infectious burden.

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3. Marco-Ayala, J. et al. TCT (2023)
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Antifungal/antiviral/anti-bacterial prophylaxis update: who, what, how long?

Speaker: *Dionysios Neofytos (Geneva, Switzerland)*

Dr Neofytos gave an update on post-transplant prophylaxis of antifungal, antiviral and antibiotic prophylaxis. When showing his disclosure slide, he joked; “The title of my presentation is who, what and how long? My other disclosure is that, unfortunately, I will not be able to answer it unequivocally. However, I like to use the opportunity to reflect on our practices and to see what we can do better.”

Risk for infections depend on timing post-transplant, engraftment and post-engraftment complications. Guidelines are helpful to define the patients at risk, and when they are at risk, what the suitable prophylactic options are, for which infections, and when to begin. However, there is limited data when to stop different types of prophylaxis.

Dr. Neofytos used a survey recently published by the EBMT IDWP, to which 127 of 553 institutions (23%) responded. The survey showed that there is a lot of variation in prophylactic strategies. The presentation focused on bacterial, HSV/VZV, CMV and PCP prophylaxis. For the purpose of this report, we’ll focus on the information he shared regarding CMV.

For CMV prophylaxis, Neofytos referred to the ECIL guideline, that previously has been extensively discussed by Prof. Ljungman (Table 1).[1] For primary prophylaxis it is recommended to start within 28 days after transplant, and continue to minimum +100 days.

Although the field of CMV has been well established, a lot of questions remain to be answered. “We still not know exactly whether we should give prophylaxis for 100 or 200 days, or even what is the exact moment to start after transplant. When we look at available data we know it is started within the first four weeks, but is it better to start at day 7, day 14 or so on, we don’t know. Also, additional factors come into play, such as newly identified risk factors as pre-transplant CMV IgG titers or pre-transplant CMV DNAemia. Or specific patient populations as T-cell depleted or haploidentical donors, for who prophylaxis perhaps should be tailored. And finally, how are we going to incorporate CMV T-cell immunity into this algorithm remains to be answered.”

Dr. Neofytos concluded that there are good approaches and agents available. However, questions remain unanswered and it is unlikely that new prospective trials will be performed to answer those. So it remains important to stay vigilant and work together to see what is the best we can do with the available data to do best for our patients. When to stop prophylaxis remains a patient-by-patient decision. Collaborative efforts are needed to identify gaps and provide realistic solutions. Vaccination strategies in the future will affect prophylactic patterns and lastly, surrogate markers of immunosuppression may provide additional support. Dr. de la Cámara adds: “You have not answered all our questions, but you have definitively highlighted all the problems.”

References:
1. Ljungman, P. et al. Lancet Infect Dis (2025)

Table 1: Adapted from Ljungman, P. et al. 2025

Recommendations for antiviral prophylaxis in adults and children

	ESCMID grade	
	Adults	Children
Letermovir is recommended as the strategy of choice for preventing CMV for CMV primary prophylaxis for CMV seropositive allogeneic HCT recipients	AI	BIIa
Letermovir prophylaxis should be started as early as feasible after allogeneic HCT to reduce the risk of early reactivations	BII	No data
Letermovir should be started no later than day 28 after transplantation	AI	No data
Prophylaxis should be continued for at least 100 days after HCT	AI	AIIu
Extended prophylaxis should be considered in patients at high risk for CMV disease and can continue to at least 200 days after transplantation	BI	CIII
For some individuals, prophylaxis for longer than 200 days after transplantation can be considered if the treating physician's judgement is that the benefit is stronger than the risk	CII	CIII
Drug-drug interactions should be considered when giving letermovir prophylaxis	BII	CIIt
Letermovir blips (single test low-level DNA positivity in plasma or whole blood samples occurring especially early during letermovir prophylaxis) are common; interruption of letermovir prophylaxis is not recommended unless there are repeated positive samples showing increased viral load	BII	CIIt
Primary letermovir prophylaxis in patients with CMV negative status, regardless of the donor serostatus, is not recommended	DII	DIII
After discontinuation of letermovir prophylaxis, secondary prophylaxis with letermovir can be considered		
After successful treatment (negative QNAT test) of a CMV reactivation in patients perceived to be at increased risk for CMV disease	BII	CIII
In patients who, for some reason, have not received primary prophylaxis and have reactivated CMV that has been successfully treated	BII	CIII
Prophylactic valaciclovir could be used if letermovir prophylaxis is not used as primary prophylaxis for CMV seropositive allogeneic HCT recipients	CI	CIIt
The use of valganciclovir, ganciclovir, intravenous immunoglobulin, or foscarnet as prophylaxis against CMV reactivation is generally not recommended	DII	CIIt (GCV),DIIt (FOS), DIII (IVIG)

ESCMID=European Society for Clinical Microbiology and Infectious Diseases. CMV=cytomegalovirus. HCT=haematopoietic cell transplant. QNAT=quantitative nucleic acid testing. GCV=ganciclovir. FOS=foscarnet. IVIG=intravenous immunoglobulin.

OS09 - Oral Session 9 | Infections - Viral
March 24th

Chairs: *Rafael de la Cámara (Madrid, Spain), Claire Horgan (Manchester, United Kingdom of Great Britain and Northern Ireland (the))*

Cytomegalovirus cell-mediated immunity guide letermovir prophylaxis after allogeneic hematopoeietic stem cell transplantation: an open label, multicenter, randomized, phase 3 trial

Speaker: *Xiaoxia Hu (Shanghai, China)*

CMV cell mediated immunity recovery is time dependent.[1] "The higher CMI, the lower risk to have a CMV reactivation."[2] Wang said.
Last year during EBMT in Florence, data on their retrospective analysis showed that CMV-CMI guided letermovir prophylaxis reduced late onset cs-CMVi.[3] In this study, the incidence of csCMVi was 13.3% in the guided group, and 28.3% in the fixed duration group. The cumulative incidence of late onset csCMVi, measured as csCMVi after letermovir withdrawal, was 9.7% in the CMV-CMI guided group vs 24.8% in the fixed duration group. Based on these results, the authors designed a prospective open-label RCT to investigate late onset csCMVi after aHSCT CMV-CMI guided letermovir prophylaxis versus fixed duration prophylaxis.[4]

The primary endpoint was the incidence of late onset csCVMi 1 year after transplantation. Secondary endpoints were CMV-associated outcomes. The researchers included 258 patients from 7 centers across China, which were randomized to 125 in the CMV-CMI-guided group and 125 in the fixed duration group. CMV-CMI was measured with ELISpot for CMV-specific T-cells that secrete IFN-y. 81.2% of the included patients received HSCT from a haploidentical donor. 82.4% of the patients received ATG based prophylaxis for GVHD.
The median follow up was 431 days.

The researchers found a significant difference at the primary endpoint: the cumulative incidence of csCMVi was 11.2% in the study group, compared to 26.2% in the fixed duration group (p=0.006). The difference remained significant until 1 year post transplant. Table 2 shows CMV-associated outcomes.

The researchers analyzed several characteristics to identify both risk- and protective factors for late onset infections. Steroid refractory GVHD was found to be a risk factor for late onset infections. CMV-CMI guided prophylaxis and the use of a myeloablative regimen demonstrated protective factors. The full summary is reflected in table 3.

The safety and tolerability were comparable in both groups. One patient discontinued letermovir due to severe vomiting. Of special interest were the incidences of HHV-6 and EBV infections. These respective incidences for HHV-6 were 8% in the CMV-CMI guided group vs 8% in the fixed duration group, and EBV occurred in 21.6% of patients vs 23.2%respectively. No significant differences were observed.

In summary, CMV-CMI guided prophylaxis showed a manageable safety profile and effective to reduce the cumulative incidence of csCMVi. Multivariable analysis showed steroid resistant aGVHD was a high-risk factor for late-onset cs-CVMi, whereas CMV-CMI guided prophylaxis and a myeloablative regimen were protective factors.

In the discussion a question was raised if the ~10% CMVi in patients in which letermovir was stopped because of positive cell-mediated immunity may have to do with the fact that they were mainly haplo-identical transplants. Hu answered that this is possible because these patients may have more immune events after 100-200 days posttransplant.

CVM associated outcomes

Characteristics	Fixed duration group (n = 117)	CMV-CMI guided group (n = 115)	p
Letermovir withdrawal, median day after allo-HCT, (range)	100	104 (100-270)	<0.001
CMV breakthrough, n (%)			
From d1 to d100	1(0.8)	4(3.4)	0.36
From d100 to letermovir withdrawal	0	4(3.4)	0.12
R/R cs-CMVi, n (%)	3 (2.5)	2(1.6)	0.21
CMV UL56 mutation, n (%)	1(0.8)	1(0.8)	0.48
CMV disease, n (%)	4 (3.3%)	0	0.42
Time from graft infusion to late-onset cs-CMVi, median (range), days	151 (134-306)	199 (113-248)	0.01
Time from letermovir withdrawal to late-onset cs-CMVi, median (range), days	49 (13-148)	45 (21-102)	0.72

Table 2:
Adapted from Wang et al. NCT06449586

Risk factors for late-onset cs-CMVi

High-risk factors: SR aGvHD; Protective factors: CMV-CMI guided Prophylaxis, Myeloablative regimen

	Univariable		Multivariable	
	HR (95%CI)	P	HR (95%CI)	P
Prophylaxis strategy				
Fised-duration	Reference		Reference	
CMV-CMI guided	0.39 (0.19–0.77)	0.007	0.45 (0.22–0.90)	0.025
HLA matching				
MSD	Reference		Reference	
HID	2.728 (0.375–19.665)	0.319		
MUD	3.753 (0.458–30.067)	0.213		
Disease status				
CR	Reference		Reference	
Active Disease	1.244 (0.554–2.505)	0.563		
Conditioning intensity				
Reduced-Intensity	Reference		Reference	
Myeloablative	0.35 (0.16–0.75)	0.007	0.37 (0.15–0.93)	0.035
SR-GvHD				
No	Reference		Reference	
Yes	2.69 (1.19–6.05)	0.017	2.99 (1.28–6.97)	0.011
EBV reactivation				
No	Reference		Reference	
Yes	0.91 (0.44–1.90)	0.25		

Table 3:
Adapted from Wang et al. NCT06449586

Reference:
1. Fu, W. et al. Best Practice & Research Clinical Haematology (2025)
2. Mele, D. et al. Bone Marrow Transplantation (2026)
3. Wang, L. et al. Clin Infect Dis. 2025
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Impact of CMV infection in patients with lymphoma after CAR-T therapy. An international study on behalf of IDWP, LWP, and CTIWP of EBMT

Speaker: *Rafael de la Cámara (Madrid, Spain)*

In this session dr. de la Camara discussed the impact of CMV infection on non-relapse mortality and survival after CAR-T therapy in lymphoma patients.

One leading cause of failure in CAR-T therapy for lymphoma patients is relapse or progression of the underlying disease (85-95%).[1] NRM rates lie between 6-10% at 1 year, with infections being the leading cause, representing approximately half of the non-relapse deaths.[2] CMV is an early and frequent infection after CAR-T in 17-56% (median 29% patients), including clinically significant cases (7-15%, median 11%), occurring 14-21 days after infusion.[3]

The impact of CMV on non-relapse mortality and survival remains largely unexplored. Three retrospective smaller studies found a correlation between CMV infection and increased mortality, one of those saw an increase in relapse as well.[4-6]

Dr. de la Camara presented a retrospective EBMT registry study. Adult lymphoma patients were included who received their first CAR-T infusion between 2019-2024 and had minimal 1 CMV PCR test performed. CMV PCR monitoring was not standardized and frequency changed between centers. The primary endpoint was the impact of CMV infection on NRM 1 year after transplantation. Because most CMVi occurs early after infusion, a 50-day landmark analysis was performed comparing outcomes of patients who developed CMVi by day 50 and those who did not.

Of the 7770 patients in the registry, 1429 patients were included (18,3%). CMV infection occurred in 205 patients (14.3%). Median time after CAR-T was 23 days 90-1282), 73% occurred within 50 days after infusion. The cumulative incidence at 100 days was 10% (95% CI 9-12), and was higher in CMV seropositive vs seronegative patients (19% vs 1%, p<0.001). Common toxicities with CAR-T therapy as Cytokine Release Syndrome (CRS) and Immune Effector Cell-Associated Neurotoxicity Syndrome (ICANS) had a cumulative incidence of 81 and 46% respectively.

The outcomes at 12 months after CAR-T in all patients was 69% overall survival (95% CI 66-71%), relapse/progression in 40% (95% CI 32-49%) and non-relapse mortality in 8% (95% CI 7-10%). There was no change in overall survival between the 2 groups, though the authors noticed differences in transplant related mortality. This was increased in the study group. CMV infection was associated with lower overall survival (p<0.001), higher NRM (40% vs 46% P<0.001) and higher relapse/progression rate (p=0.043).

Tables 4 and 5 show a summary of key results and key risk factors.

Table 4: Adapted from De la Cámara, R. et al.

Patients included in the CVM study (study group) vd rest of CAR patients

Outcomes (at 12 months post CAR T)	Study group vs rest of CAR T	Outcome over the years (2019-2024)	
		Study group (n= 1429)	Rest of CAR T (n= 6341)
Overall survival	No differences 69% vs 67% p.11	No change (76-67%) p .16	Increased (59-71%) P <0,001
Relapse/progression	40% vs 46% p <.001	No change (48-41-%) p .6	Decreased (60% to 40%) p <0,01
TRM	8% VS 7% p.005	Increased (2-11%) p .005	No change (6-8%)

Table 5: Adapted from De la Cámara, R. et al.

Multivariate cox model

CMV infection was associated

Lower overall survival: HR 2.11; 95%CI 1.65, 2.70; p <.001

Higher NRM: HR 2.51; 95%CI 1.56, 4.01; p <.001

Higher Relapse/progression: HR 1.36; 95% CI 1.01-1.84; p =.043

Cox model

Variables, N=1351	HR	95% CI	p-valor
CMV	**2.51**	**1.56, 4.01**	**<0.001**
Sex			
Female	-		
Male	1.50	1.06, 2.12	0.023
Year of therapy			
2019/2020	-		
2021	2.30	1.17, 4.50	0.016
2022	2.17	1.05, 4.37	0.031
2023	2.71	1.35, 5.43	0.005
2024	3.17	1.46, 6.59	0.004
Age at treatment	1.53	1.14, 1.56	<0.001
Disease stage at CT			
CR + ?R	-		
Rel/prog	1.42	1.00, 2.03	0.050
Stable	0.94	0.47, 1.89	0.9
NA	1.83	0.79, 4.27	0.2

Variables, N=1353	HR	95% CI	p-valce
CAR-T product			
Yescarta	-	-	
Brevani	1.99	0.82, 4.83	0.13
Kymriah	0.59	0.35, 1.01	0.053
Tecartes	1.72	1.12, 2.64	0.013
Previous transplant			
No	-	-	
Yea	0.98	0.67, 1.42	>0.9
ECOG score			
ECOG 0-1	-	-	
ECOG 2+	3.56	2.28, 5.55	<0.001
Missing	1.33	0.65, 2.69	0.4
frailty (treatment clc)			0.023

When looking specifically into the 50-day landmark analysis, the researchers saw patients that developed a CMV infection within the first 50 days post-transplant had worse outcomes and lower overall survival (62% vs 74%, p=0.003). However, CMV infection <50 days was not associated with relapse/progression or NRM.

The take home messages of the study were:

- This was the largest series of CMVi reported in CAR-T lymphoma patients, though it represented a small proportion of all CAR-T during the study period (18.3%)
- CMVi was higher in CMV seropositive patients and was an early complication
- CMVi was associated with worse outcomes, namely lower overall survival higher relapse/progression and higher NRM. Multvariate landmark analysis at 50 days showed lower overall survival was associated with early infection

This study is not sufficient to determine if CMVi is a cause of the worse outcomes or a marker in high risk patients. More studies are necessary.

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Intravenous brincidofovir effectively reduces CMV DNAemia in antiviral experienced immunocompromised patients: results of a phase 2A clinical trial

Speaker: *Genofeva A. Papanicolaou*

Although we have come a long way with CMV prevention and treatment, reducing the mortality and morbidity associated with CMV. Nonetheless, currently available CMV-specific antivirals are associated with toxicities and/or the rapid development of resistance." started Papanicolaou her presentation. "As such, there remains an unmet need for safer and more effective antiviral options in immunocompromised patients".

She showed the results of the Athena phase 2a clinical trial[1], introducing intravenous brincidofovir as a lipid conjugate of cidofovir, which allows for more efficient cellular penetration and higher intracellular drug concentrations. This results in a >100-fold increased potency against CMV compared to cidofovir, while maintaining the same mechanism of action. Importantly, brincidofovir is not a substrate for human OAT1 transporters, which are responsible for renal accumulation of cidofovir, and is therefore not associated with nephrotoxicity. She also noted that the dose-limiting gastrointestinal toxicity observed with oral brincidofovir may be reduced with intravenous administration.

The objectives of the study were to evaluate the safety and tolerability of intravenous brincidofovir as treatment for CMV in immunocompromised patients, as well as its antiviral efficacy, assessed by changes in CMV DNAemia from baseline during treatment.

Dr. Papanicolaou continued by describing the study design, which consisted of a descending dose study with two cohorts. Patients in cohort A received IV brincidofovir 0.4 mg/kg twice weekly (BIW) (n=6 planned; n=9 enrolled), while patients in cohort B received 0.5 mg/kg BIW (n=6 planned; n=10 enrolled). Treatment was administered for up to 8 weeks or until viral clearance plus an additional 2 weeks, with follow-up extending to 30 days after the last dose. Key inclusion criteria included male or female, age ≥2 months, CMV viremia ≥500 IU/mL with or without symptomatic CMV disease, and an underlying immunocompromised state. Exclusion criteria included subjects who weigh >120 kg, and patients with known toxicities of the oral formulation. Baseline patient characteristics are shown in table 6.

	Cohort A (IV BCV 0.4 mg/kg); n=9	Cohort B (IV BCV 0.5mg/kg);n=10
Mean age, years (range)		
Pediatric (less than 18 years of age) (%)	3 (33)	5 (50)
Adult (18 years of age and older)	6 (67)	5 (50)
Underlying immunocompromising conditions,		
HSCT/SOT/Others	6/1/2	7/2/1
Plasma CMV DNA baseline central laboratory, n (%)		
Very low (≥455 to <910 IU/mL)	2 (22)	3 (30)
Low (≥910 to <9100 IU/mL)	3 (33)	5 (50)
High (≥9100 IU/mL)	4 (45)	2 (20)
Mean baseline by Cohort	3.99 log10 IU/mL	3.39 log10 IU/mL
Use of T-cell depletion therapy, n (%)		
T-cell depletion	4 (67)	3 (50)
No T-cell depletion	2 (33)	3 (50)
History of CMV prophylaxis, n (%)		
Refractory/Resistant	7 (78%)	7 (70)
Preemptive	2 (22)	3 (30)

Table 6: Adapted from Athena phase 2a Intravenous Brincidofovir

She then presented patient disposition data, noting that 7 out of 9 patients in each cohort had failed at least one prior CMV antiviral therapy (ganciclovir/valganciclovir n=12, foscarnet n=4, letermovir n=4, maribavir n=2). All patients received more than 1 week of brincidofovir, and the majority received more than 4 weeks of treatment (8/9 in cohort A and 6/9 in cohort B).

With regard to efficacy, intravenous brincidofovir effectively reduced CMV viral load. At week 4, CMV DNA decreased to below the lower limit of quantification (LLOQ; 2.0 log10 IU/mL) in 2/9 patients (22%) in cohort A and 5/9 patients (56%) in cohort B. The mean decline in CMV DNA at week 4 was −1.33 log10 IU/mL and −1.26 log10 IU/mL, respectively. At week 8, CMV DNA was below LLOQ in 4/9 patients (44%) in cohort A and 8/9 patients (89%) in cohort B, with mean declines of −1.67 log10 IU/mL and −1.80 log10 IU/mL, respectively.

In terms of safety, most patients experienced TAEs, occurring in 78% of patients in cohort A and 70% in cohort B. "Drug-related AEs included mild diarrhea for one patient in each cohort, elevated liver enzymes in two patients in cohort B, and one allergic reaction." according to Papanicolaou. "These events led to the discontinuation of brincidofovir. Two deaths occurred in cohort A, both considered unrelated to the study drug."

In conclusion, Dr. Papanicolaou stated that intravenous brincidofovir demonstrated antiviral activity in reducing CMV viremia and was reasonably well tolerated in pre-treated immunocompromised patients. The higher dose of 0.5 mg/kg showed a trend towards greater antiviral efficacy, but was also associated with a higher rate of drug-related adverse events. These findings support further clinical development of intravenous brincidofovir for the treatment of CMV infection.

Maribavir for clinically significant cytomegalovirus infection in allogeneic hematopoietic cell transplantation: a real-world multicenter study of IDWP

Speaker: *Annalisa Paviglianiti (Barcelona, Spain)*

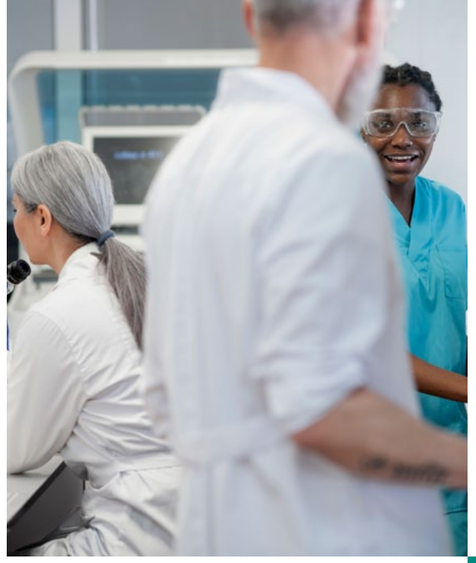
Maribavir has been approved by the EMA since November 2022 for the treatment of resistant/refractory CMV infection or disease in adult allogeneic HSCT or SOT recipients. Dr. Paviglianiti noted its favorable toxicity profile, although resistance develops in approximately 7–10% of patients. However, real-world experience remains limited.

The aim of the retrospective international real world study of the IDWP of EBMT was to report clinical experience with maribavir in the HCT setting.[1] Primary endpoint was CMV resolution at 12 weeks after maribavir start, secondary endpoints included reasons for discontinuation, overall survival and non-relapse mortality at 12 weeks after maribavir start.

This retrospective EBMT registry included 118 patients from 37 centers (transplanted between March 2021 and October 2024), who received 126 maribavir courses between December 2022 and January 2025. Both adult and pediatric patients were included, with a minimum follow-up of 12 weeks. The main indication was clinically significant CMV infection.

Of the 118 patients, 90% (n=106) were adults and 8% (n=9) pediatric. Most patients were male (63%, n=74), and acute leukemia was the most common underlying disease (53%, n=61). The majority received ATG (84%, n=99), underwent haploidentical transplantation (36%, n=42), and had PTCy-based GVHD prophylaxis (54%, n=64). Letermovir prophylaxis had been used in 53% (n=63), and donor–recipient serostatus was R+/D+ in 50% (n=58), 39% (n=45) were R+/D-. Prior acute GVHD grade 2–4 was reported in 36% (n=41), and moderate-to-severe chronic GVHD in 9% (n=10).

Dr. Pavglianiti next addressed treatment characteristics. The median time from HCT to first maribavir course was 18.6 weeks, with a median treatment duration of 7.9 weeks. Maribavir was mainly used as second-line PET (48%, n=61), but also in first-line (19%, n=24) and later lines (19%, n=24), as well as for CMV disease. The main reasons for use were refractory/resistant CMV (52%, n=64) and toxicity of prior therapies (48%, n=62). Resistance testing prior to treatment was performed in 6% of courses (n=8), identifying mutations in UL54, UL97, and UL56.



Among the 109 PET courses, CMV resolution was achieved in 81% (n=88), while response was not evaluable in 2% (n=2). Treatment failure occurred in 17% (n=19), including progression in 7% (n=8), response with rebound in 9% (n=10), and no response in 1% (n=1).
Subsequently, outcomes in patients with CMV end-organ disease were reported. Seventeen courses were administered for CMV end-organ disease, including gastrointestinal disease (71%, n=12), gastrointestinal disease with pneumonia (6%, n=1), pneumonia (12%, n=2), skin disease (6%, n=1), and multi-organ disease (6%, n=1). Response was observed in 70% (n=12), progression in 12% (n=2), and response was not evaluable in 18% (n=3).

Most treatment courses were completed (76%, n=90). Discontinuation occurred due to death (11%, n=13), response with rebound (7%, n=9), adverse events (2%, n=2), supply issues (2%, n=2), or other reasons (2%, n=2). During PET, CMV end-organ disease developed in 5% (n=5) of cases.
In the pediatric subgroup (n=9), two patients (22%, n=2) had received letermovir prophylaxis. Response to maribavir was observed in 63% (n=5/8) of PET courses and 50% (n=1/2) of CMV disease cases. In the overall cohort, 12 week OS was 82% (95% CI 75–89%) and NRM was 15% (95% CI 9–22%).

In conclusion, Dr. Pavglianiti stated that this large real-world cohort confirms maribavir as an effective treatment option for clinically significant CMV infection in the HCT setting, with high response rates and limited toxicity. She noted that use beyond EMA-approved indications appears promising and warrants further investigation.

In response to a question from the audience regarding resistance development, Dr. Pavglianiti noted that these data were only available for 60 treatment courses. Based on these limited data, resistance appeared to be low.

OS10 - Oral Session 10 | Stem cell donor and source
March 24th

Chairs: *Alessandro Criscimanna (Milano, Italy), Rocio Parody (Jerez de la Frontera, Spain)*

Letermovir eliminates historic CMV-related outcome differences in MUD allo-SCT

Speaker: *Radwan Massoud (Hamburg, Germany)*

Historically, CMV seropositivity increased NRM after aHSCT. Donor/recipient matching stratified patient outcomes.[1] The German single centre study presented, aimed to see if these differences persist after routine letermovir prophylaxis.

The researchers included 1353 patients, n=1062 in the pre-letermovir era and n=291 in the post-letermovir era. The pre-letermovir era was further divided in early (2010-2015, n=445) and late era (2016-2021, n=617). All CMV donor/recipients pairings were analyzed (R+/D+, R+/D-, R-/D+, R-/D-).

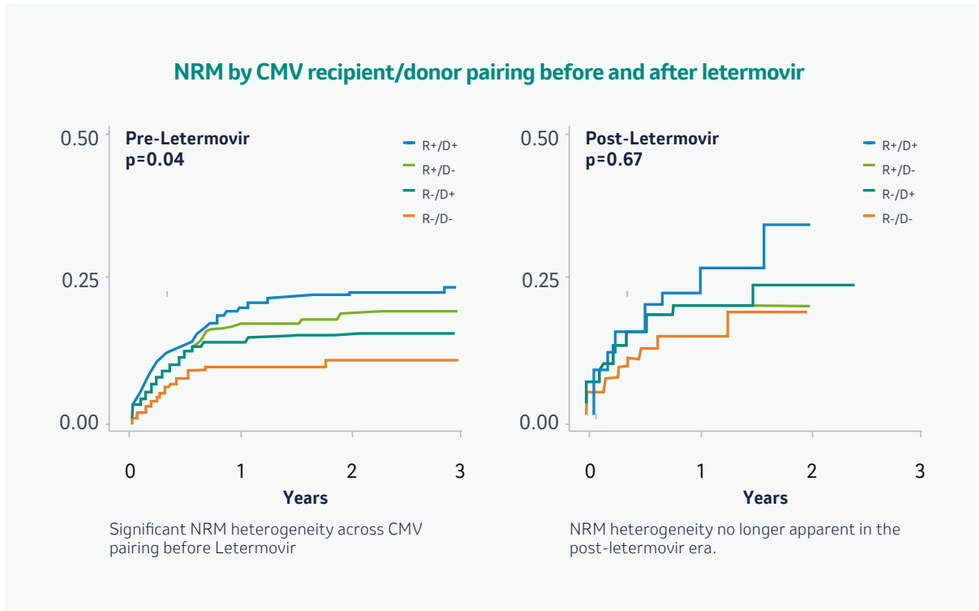
Non-relapse mortality, relapse, overall survival and progression free survival were assessed. Overall the cohorts were well balanced and reflected a large real world matched unrelated transplant population. There were some misbalances and this was adjusted for that in the multivariable analysis.

Non-relapse mortality was significantly associated with a positive CMV serostatus in the pre-letermovir era (p=0.01 in the early era, p=0.08 in the late era respectively). This difference was not observed in the post-letermovir era (p=0.9). The results were tested in a multivariate analysis, in which the overall results were confirmed. Based on these results, the researchers concluded that letermovir eliminates CMV D/R pairing differences across all major transplant outcomes (NRM, OS, PFS and relapse) (Figure 8).

These findings suggest reduced contemporary relevance of CMV D/R matching in MUD donor selection.

References:
1. Schmidt-Hieber, M. et al. Blood (2013)

Figure 8: Adapted from Radwan Massoud, EBMT 2026



Significant NRM heterogeneity across CMV pairing before Letermovir

NRM heterogeneity no longer apparent in the post-letermovir era.

OS18 - Oral Session 18 | CAR-T and other Cellular Therapies March 25th

Chairs: *Margot Jak (Utrecht, Netherlands the), Aibin Liang (Shanghai, China)*

Adoptive transfer of multivirus specific T cells against refractory CMV, ADV and EBV infections after stem cell transplantation – interim results from the "TRACE" phase-III trial

Speaker: *Theresa Kaeuferle (Freiburg, Germany)*

Dr. Kaeuferle introduced the clinical challenge of chemotherapy-refractory CMV, EBV, or AdV infections following aHSCT. These patients are exposed to high morbidity and mortality, while treatment options remain limited and T-cell reconstitution may take up to 6 months post-transplant.[1,2] She noted that adoptive transfer of multivirus-specific T cells has shown promising results in early studies. The aim of this phase III trial was therefore to restore antiviral immunity through infusion of virus-specific T cells.[1]

In this treatment approach, peripheral blood mononuclear cells (PBMCs) are isolated from the original HSCT donor and stimulated with CMV-, EBV-, and AdV-specific peptides. Interferon-γ–secreting cells are subsequently selected using a cytokine capture system. These multivirus-specific T cells are then infused into the patient, where viral antigens drive in vivo expansion of these cells, leading to sustained antiviral immune protection.[3]

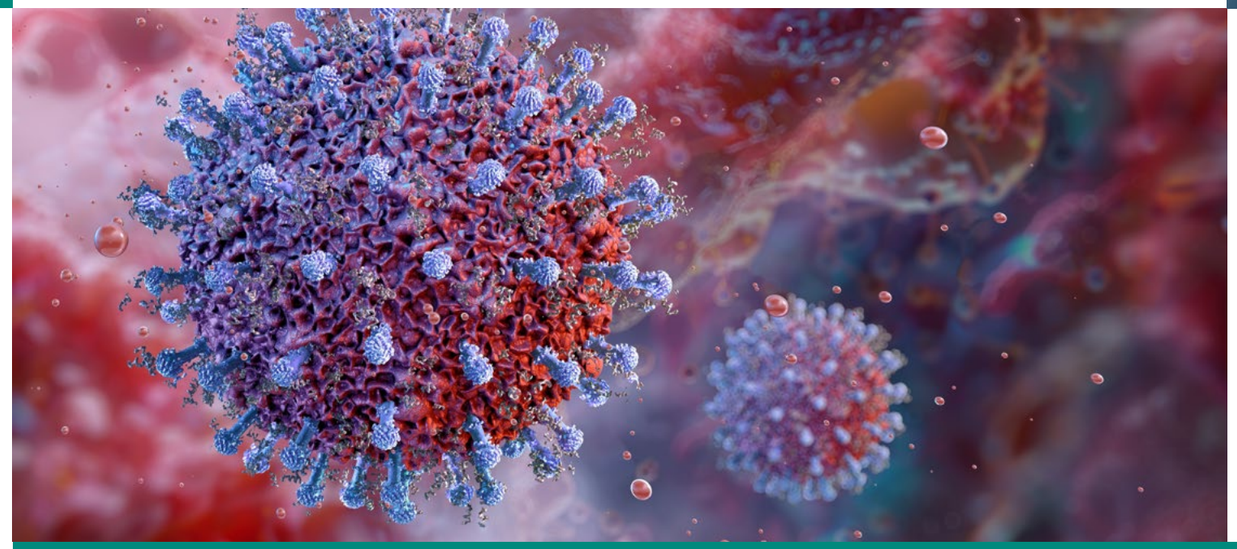
The TRACE study is a multicentre, double-blind, placebo-controlled, randomized phase III trial conducted across 35 centres in 6 European countries. Patients are randomized in a 2:1 ratio to receive T-cell therapy or placebo. The primary endpoints are viral clearance and viral progression. Kaeuferle first presented the interim safety analysis. Acute GVHD (grade III/IV) was defined as a key adverse event of special interest. Patients were divided into four subcohorts of approximately 15 patients each. Not all patients have been evaluated, but to date only one case of acute GVHD was reported. The observed incidence was well below predefined critical thresholds, and no stopping rules were met. No acute toxicities had been reported apart from GVHD. The Data Safety Monitoring Board reviewed all safety data and confirmed the absence of safety concerns, recommending continuation of the study.

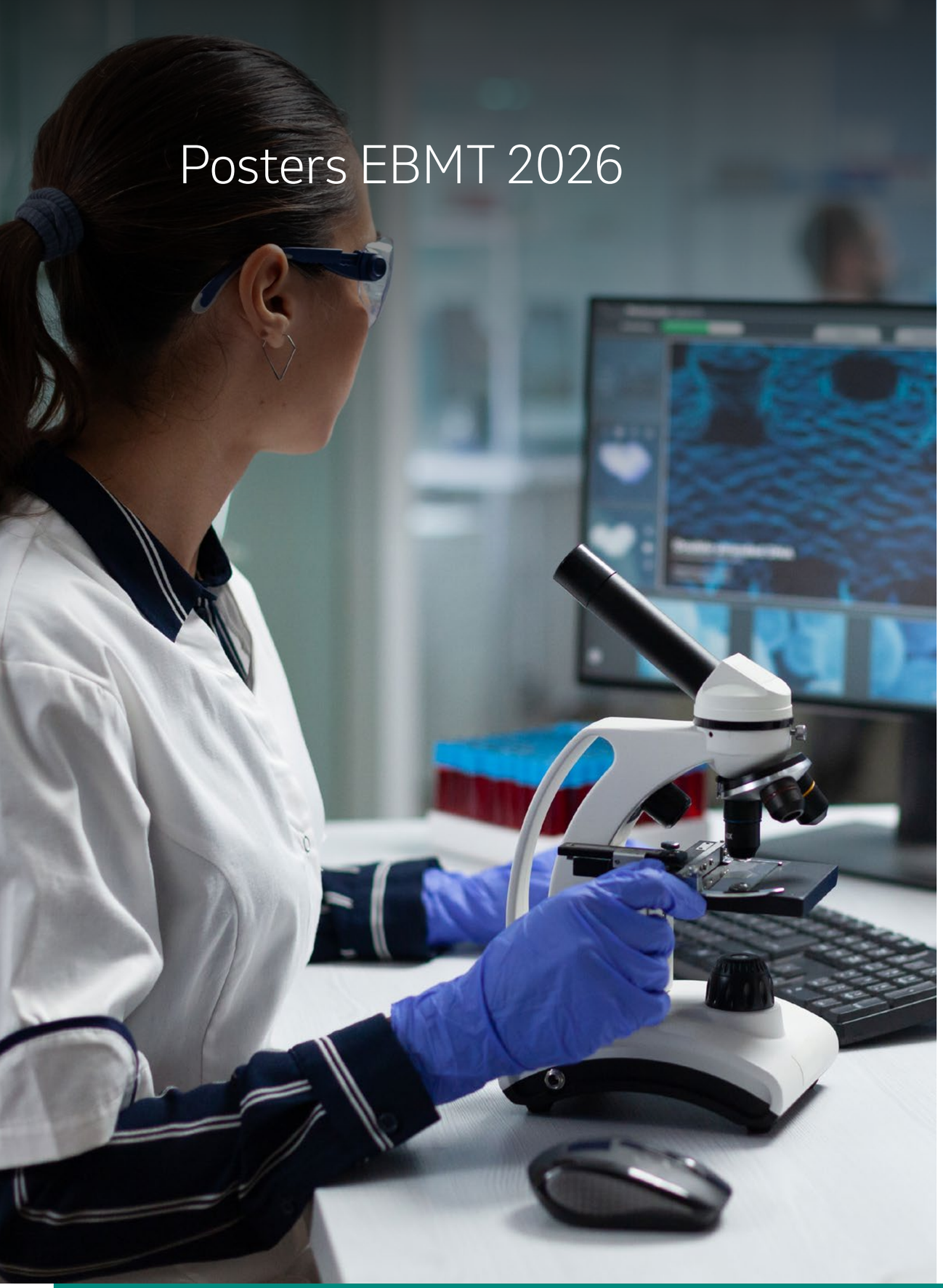
Efficacy interim results were then presented for 39 evaluable patients. For the first primary endpoint, viral clearance at week 8 was achieved in 56% of patients in the T-cell group compared with 36% in the placebo group. For the second primary endpoint, absence of viral progression at week 8 was observed in 76% versus 57% of patients, respectively.

Although numerical differences were observed for both endpoints, these were not statistically significant.

Dr. Kaeuferle emphasized that, given the limited sample size, no definitive conclusions can be drawn at this stage, and the trial will continue as planned.

References:
1. Feuchtinger, T. et al. Leukemia & Lymphoma (2006)
2. Yong, K.Y. et al. Open Forum Infect Dis (2025)
3. Feuchtinger, T. et al. J Hematol Oncol (2019)





Posters EBMT 2026

This section gives an overview of the posters related to cytomegalovirus (CMV) presented at the 52nd Annual Meeting of the EBMT. They are subdivided in the following categories:

1. Real world evidence (Efficacy & safety, Special populations, Late CMV infection)
2. Co-infection (including EBV)
3. Refractory/resistant CMV infection
4. Novel therapeutic & prophylactic approaches
5. State of the art: diagnostics, monitoring & immune reconstitution
6. Case reports

1. Real World Evidence: Efficacy & Safety

OUTCOME OF CMV PNEUMONIA AFTER HEMATOPOIETIC CELL TRANSPLANTATION IN THE ERA OF MODERN TRANSPLANTATION AND CMV PREVENTION PRACTICES

Alicja Sadowska-Klasa *Fred Hutchinson Cancer Center, Seattle & Medical University of Gdansk, Gdansk, Poland*
EBMT 2026, Poster B084

Routine PCR surveillance and the introduction of letermovir prophylaxis have sharply reduced the incidence of early CMV pneumonia after allogeneic HSCT, but late CMV disease still occurs and contributes to transplant-related deaths. This retrospective study reviewed proven or probable CMV pneumonia cases to assess current CMV-pneumonia related mortality and to examine whether antivirals and immunoglobulin administration improve patient outcomes.
The authors retrospectively identified 448 cases of proven or probable CMV pneumonia from 1986–2022; though numbers fell over time (133 cases in 2000–2011, 26 cases in 2012–2022). A focused analysis of 141 patients treated with antivirals (in post 2000 transplants) assessed recipient, donor, transplant, viral and treatment-related factors that were linked to overall mortality or attributable to CMV.

The key findings of the study were:

- CMV-related mortality has declined significantly in the modern era, reaching 11% (95% CI 2.8–27.1%) in the most recent period
- In univariable analysis, female sex, lymphopenia, elevated bilirubin before CMV onset, need for mechanical ventilation, high-dose steroids (>2 mg/kg), and diagnosis of DAH/IPS were associated higher overall and CMV-attributable mortality. After multivariable adjustment, only mechanical ventilation, elevated bilirubin, and female sex remained independent risk factors
- The proportion of patients needing mechanical ventilation at diagnosis stayed high (26.3% in 2000–2011 vs. 50% in 2012–2022)
- Adding IVIG or CMV-specific immunoglobulin was not linked to improved overall survival compared with antivirals alone
- Survival for ventilated patients improved in the recent era, likely reflecting advances in supportive care and ventilation strategies rather than changes in antiviral regimens

CMV pneumonia incidence and mortality have continued to decrease in the modern era, although late CMV episodes remain relevant. Adding immunoglobulin showed no clear survival benefit, and outcomes for ventilated patients—though improving—remain poor, highlighting the need for continued focus on prevention, early detection, and optimized supportive care.

THE IMPACT OF CMV-SEROPOSITIVITY ON OUTCOMES AFTER ALLOGENEIC STEM CELL TRANSPLANTATION IN THE ERA OF LETERMOVIR IN AML AND MDS PATIENTS

Lina Kolloch, *University hospital Münster Münster, Germany*
EBMT 2026, Poster B094

CMV serostatus has historically been associated with survival outcomes and relapse incidence after aHSCT, but its impact in the era of letermovir prophylaxis remains unclear. This single-centre retrospective study evaluated the association between patient and donor CMV serostatus and key clinical outcomes, including survival, relapse, and GVHD.
A total of 548 patients with AML or MDS undergoing allo-SCT (2018–2024) were included, of whom 61% (n=333) were CMV seropositive and 39% (n=215) CMV seronegative. The majority of CMV seropositive patients received letermovir prophylaxis (97%). Baseline characteristics were well balanced between groups.
At a median follow-up of 2 years for the whole cohort, CMV seronegative patients had significantly better relapse-free survival (73% vs 66%, p=0.026) and a trend towards improved overall survival (76% vs 74%, p=0.071). Relapse incidence was numerically lower in CMV seronegative patients (13% vs 20%, p=0.075), while non-relapse mortality was comparable between groups.

Within the CMV seropositive cohort, donor serostatus appeared relevant, with a trend towards higher relapse incidence in patients transplanted from CMV seropositive donors (23% vs 12%, p=0.056), but significantly higher non-relapse mortality in patients with CMV seronegative donors. CMV reactivation requiring pre-emptive therapy did not impact survival outcomes. Incidence of acute and chronic GVHD and duration of immunosuppression were similar between groups. Interestingly, immune reconstitution at day +100 was faster in CMV seropositive patients, with higher CD4+ (median 137 vs 108/µL, p=0.028) and CD8+ T-cell counts (230 vs 201/µL, p=0.016).

These findings suggest that CMV serostatus may still influence outcomes after aHSCT in the letermovir era, particularly with regard to relapse-free survival, while no clear association was observed between CMV infection or its management and survival outcomes.

LETERMOVIR PRIMARY PROPHYLAXIS IS A PROTECTIVE FACTOR FOR SURVIVAL IN HEMATOPOIETIC CELL TRANSPLANT RECIPIENTS. DATA FROM THE FIRST PROSPECTIVE MULTICENTER STUDY IN LATIN AMERICA

Fabian Herrera, *CEMIC, Buenos Aires, Argentina*
EBMT 2026, Poster B097

Letermovir prophylaxis is not widely available in many Latin American countries, and real-world data on its use remain limited. This prospective multicentre observational cohort study, conducted across 9 referral centers in Argentina (2020–2025), evaluated the incidence of cs-CMVi, CMVd, CMV-related hospitalization, and mortality in adult CMV-seropositive aHSCT recipients receiving letermovir prophylaxis until day 100 or managed with PET. This is one of the first prospective studies addressing these outcomes in this setting.

In total, 551 patients were included, of whom 37% (n=205) received letermovir and 63% (n=346) were managed with PET. The letermovir group represented a higher-risk population, with older patients and a higher proportion of haploidentical transplants. In addition, a larger proportion of patients in the letermovir group were classified as high risk for CMV disease (97.1% vs 88.2%, p<0.001).

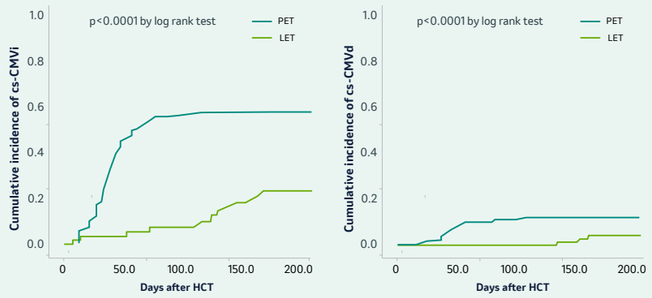
Despite this, clinically significant CMV infection (cs-CMVi) at day 100 occurred in 7.3% (n=15) versus 54.3% (n=188) in the PET group (p<0.001). At day 200, rates were 20.0% (n=41) versus 55.5% (n=192), respectively (p<0.001).

CMV disease at day 100 was observed in 0% versus 7.5% (n=26) (p=0.001), and CMV-related rehospitalization in 10.2% (n=21) versus 37.9% (n=131) (p<0.001).

Overall mortality at day 200 was numerically lower in the letermovir group (16.1% [n=33] vs 21.7% [n=75], p=0.11). In multivariable analysis, letermovir prophylaxis was independently associated with improved survival (OR 0.51, 95% CI 0.29–0.89, p=0.018), while graft failure, high-dose corticosteroids, and lymphopenia were identified as risk factors for mortality.

These findings show that letermovir prophylaxis markedly reduced CMV-related complications and rehospitalizations and was identified as a protective factor for survival in this higher-risk transplant population.

Cumulative incidence of cs-CMVi and CMVd



Adapted from F. Herrera, EBMT 2026, Poster B097

CURRENT TRENDS IN THE MANAGEMENT OF CYTOMEGALOVIRUS IN CELL THERAPIES RECIPIENTS IN SPAIN: RESULTS OF THE 2025 GETH-TC SURVEY

I. García-Cadenas, *Hospital de Sant Pau Barcelona, Spain*
EBMT 2026, Poster B117

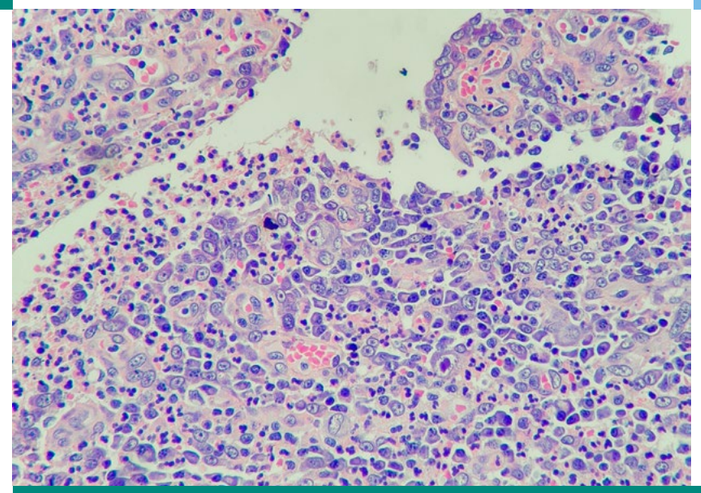
CMV management after aHSCT continues to evolve with the introduction of novel antivirals. This nationwide survey provides insight into current clinical practice across Spain. A structured online survey, distributed in May 2025 to adult and paediatric haematologists within the GETH-TC, assessed key domains including antiviral use, diagnostics, and implementation of ECIL-10 recommendations.

A total of 26 HSCT centres (65% of Spanish transplant units) participated. All centres reported the use of letermovir as primary prophylaxis, most commonly restricted to high-risk CMV-positive recipients (54%), while 23% administered letermovir to all CMV-positive patients. Among recipients receiving PTCy, 50% of centres prescribed letermovir only in the presence of additional risk factors. Prophylaxis beyond day +100 was used by 81% of centres, particularly in the presence of GVHD or delayed immune reconstitution.
Maribavir was incorporated into routine management for refractory or resistant CMV in 84% of centres. All units reported CMV monitoring protocols, although practices varied: 54% modified PET thresholds in the context of letermovir, resistance testing was available in 65%, and CMV-specific immune monitoring in 19%. No centre measured letermovir plasma levels.

Management of CMV in CAR-T and BiTE recipients remained heterogeneous. Routine CMV monitoring in CMV-positive patients was performed by 60% of centres for CAR-T and 8% for BiTE recipients, while pre-emptive therapy was commonly applied (88% and 74%, respectively). Use of letermovir or maribavir in these settings remained limited.

Regarding guideline implementation, 85% of centres planned to adopt selected ECIL-10 recommendations, particularly dual-time-point serology (65%), baseline CMV PCR testing (69%), and updated serostatus nomenclature (69%).

Overall, CMV prevention and treatment strategies are widely implemented in Spain, with broad adoption of letermovir and extension of prophylaxis beyond day +100, alongside increasing use of maribavir. However, variability persists in prophylaxis strategies, pre-emptive thresholds, and the management of patients receiving newer cellular therapies.



IMPACT OF LETERMOVIR PROPHYLAXIS ON CYTOMEGALOVIRUS EVENTS AND RELAPSE AFTER ALLOGENEIC HEMATOPOIETIC STEM CELL TRANSPLANTATION FOR AML AND MDS

Sin Young Park, *Seoul National University Hospital, Seoul, Republic of Korea*
EBMT 2026, Poster B132

Early CMV reactivation has previously been associated with a reduced risk of relapse after aHSCT, but its relevance in the letermovir era remains unclear. This single-centre retrospective study of adult patients undergoing first HSCT (2016–2024) evaluated the impact of letermovir prophylaxis on CMV events and relapse outcomes in patients with AML and MDS.

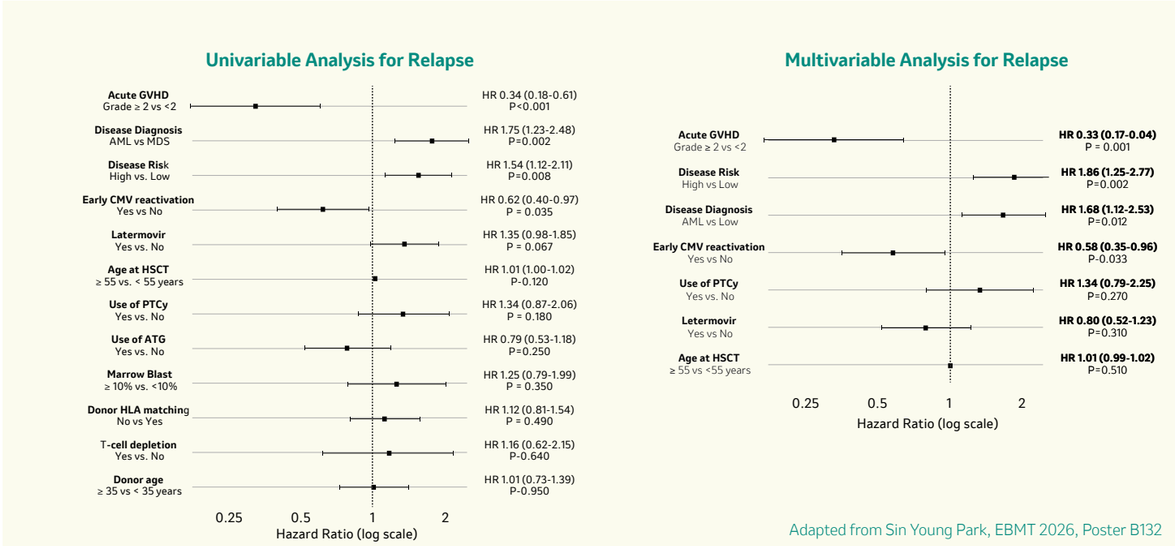
A total of 397 patients were included, of whom 43.1% (n=171, CMV-seropositive) received letermovir prophylaxis from day +7 to +100. Baseline characteristics were largely balanced, although more contemporary transplant approaches were used in the letermovir group.

Letermovir significantly reduced CMV reactivation by day +100 (6.4% vs 40.0%, p<0.001) and CMV disease (1.16% vs 8.44%, p=0.013).

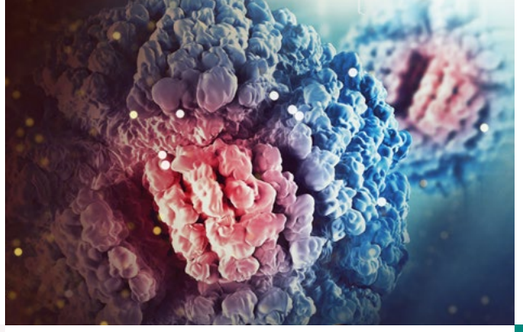
Overall survival at 2 years was comparable between groups (56.2% vs 57.1%, p=0.99). However, the cumulative incidence of relapse was higher in the letermovir group (40.0% vs 29.7%, p=0.034), while non-relapse mortality did not differ. The 100-day cumulative incidence of grade ≥2 acute GVHD was lower with letermovir (19.4% vs 28.3%, p=0.028).

The inverse association between early CMV reactivation and relapse was observed in the non-letermovir group, but not in patients receiving letermovir, likely reflecting the markedly reduced incidence of CMV reactivation. In multivariable analyses, letermovir was not independently associated with relapse, whereas early CMV reactivation remained associated with a reduced relapse risk.

These findings suggest that letermovir alters the CMV landscape after aHSCT and may obscure a CMV-related immune effect associated with graft-versus-leukemia activity, without increasing relapse risk.



Adapted from Sin Young Park, EBMT 2026, Poster B132



LETERMOVIR CYTO-MEGALOVIRUS PROPHYL-AXIS IN ALLOGENIC HEMATOPOIETIC-CELL TRANSPLANTATION: REAL-WORLD EVIDENCE FROM HIGH-ENDEMIC REGION

Sandipkumar Kheni (Poster Presenter: Velu Nair), *Apollo hospital international limited, Gandhinagar, India*
EBMT 2026, Poster B151

Real-world comparative data on letermo-vir use in mixed-donor, resource-limited transplant settings remain limited. This single-centre retrospective study from India (2019–2025) evaluated the impact of letermovir prophylaxis on CMV outcomes and survival in CMV-seropositive aHSCT recipients, following the introduction of routine prophylaxis in this center in 2024.

A total of 75 patients were included, of whom 23% (n=17) received letermovir prophylaxis and 77% (n=58) did not. Baseline characteristics were comparable between groups, although a high proportion of patients underwent haploidentical transplantation and myeloablative conditioning. Letermovir prophylaxis significantly reduced CMV-related complications. Clinically significant CMV infection by week 12 occurred in 35% versus 75.9% of patients (p=0.004), while CMV recurrence was observed in 12% versus 38.4% (p=0.03). No patient in the letermovir group developed CMV disease (0% vs 4%).

Steroid-treated GVHD was numerically lower with letermovir (17.6% vs 34.4%). Overall survival was significantly improved (94% vs 62%; p=0.01), and no transplant-related mortality was observed in the prophylaxis group (0% vs 9%).
These findings reinforce the role of letermovir as an effective and valuable addition to antiviral prophylaxis strategies for high-risk post-transplant patients.

OUTCOMES OF USING GENERIC LETERMOVIR FOR CMV PROPHYLAXIS IN HSCT: RETROSPECTIVE ANALYSIS

Anil Aribandi, *Sindhu Hospitals Hyderabad, India*
EBMT 2026, Poster P177

Real-world data on generic letermovir prophylaxis remain limited, particularly in high CMV seroprevalence settings such as India. This single-centre retrospective study from Hyderabad (March–November 2025) evaluated CMV outcomes following primary prophylaxis with generic letermovir, initiated from day +1 until day +100 post-transplant.

A total of 27 aHSCT recipients were included, with 51.8% (n=14) pediatric and 48.1% (n=13) adult patients. All donor–recipient pairs were CMV seropositive (R+/D+), and the majority underwent haploidentical transplantation (81.5%, n=22), reflecting a high-risk population with a heterogeneous range of underlying diseases.

CMV reactivation occurred in 29% (n=8) of patients, with onset between day +12 and +68 post-transplant. No cases of CMV disease or CMV-related mortality were observed, and all patients responded to first-line pre-emptive therapy.

Overall mortality was low (3.7%, n=1) and unrelated to CMV. The relatively early onset of reactivation highlights the need for close monitoring in the early post-transplant phase.

Compared with Western data, the incidence of CMV reactivation appeared slightly higher, but remained manageable with PET. These findings support the effectiveness of generic letermovir as a prophylactic strategy in high-risk transplant settings

LOW-LEVEL PRETRANSPLANT CMV DNAEMIA IS ALREADY A RISK FACTOR FOR POSTTRANSPLANT CMV INFECTION AFTER ALLOGENEIC HEMATOPOIETIC CELL TRANSPLANTATION

Catia Vieira Gomes, **Poster Presenter Dionysios Neofytos**, *Geneva University Hospital, Geneva, Switzerland*
EBMT 2026, Poster A258

Clinically significant CMV infection still occur after aHSCT, even with letermovir prophylaxis. Previously, the authors found that patients with detectable pre HSCT CMV DNAemia had a higher 180 day csCMVi rate than those with undetectable PCR (59.3% vs 26.3%).

This retrospective study further examines how pre HSCT CMV viral load (VL) affects post transplant csCMVi and hematologic outcomes.

In total 505 aHSCT patients with ≥1 CMV positive PCR (detectable (DT) or quantifiable (QT)) up to 90d prior to HSCT were included (2015-2023). Since 2019, letermovir prophylaxis was administered to CMV-R+ patients for 100d post-HSCT. CMV PCR was performed at pre-HSCT consultation, HSCT admission, day of HSCT, and as clinically indicated. Post-HSCT CMV PCR was performed weekly up to 90 days post transplant and as clinically indicated thereafter. CsCMVi was defined as CMV PCR ≥ 500 IU/mL during letermovir administration and ≥ 150 IU/mL without letermovir.

Out of the total patients, 62/505 (12.5%) patients had ≥1 positive pre HSCT CMV PCR. You find the key findings in the first table.

There was no significant association found between higher pre HSCT CMV DNAemia and csCMVi, acute GVHD, relapse, or death at thresholds of 150, 500, or 1,000 IU/mL. When looking into neutropenia and lymphopenia pre-HSCT (15/62, 24.2% and 37/62, 59.7% respectively), the researchers found only neutropenia was associated with a higher incidence of aGVHD, but no link to csCMVi, relapse or 1-year mortality.
The results are summarized in the second table.

In summary, pre-HSCT CMV DNAemia was strongly associated with increased post-HCT csCMVi incidence, regardless of the viral load. These results suggest that any DT pre-HSCT CMV DNAemia may represent an activity of CMV reservoir and could be considered as a relevant risk factor for csCMVi.

Detected (DT) versus Quantified (QT) pre-HCT CMV DNAemia

	Overall	DT (%)	QT (%)	p-value
Total number of patients	62	38 (61)	24 (39) Median: 115 IU/ml Mean: 4,392 IU/ml Range: 38.2-95,600	
CsCMVi	39 (63)	23 (61)	16 (67)	0.63
aGvHD	37 (60)	23 (61)	14 (58)	>0.05
Relapse	27 (44)	18 (47)	9 (38)	>0.05
Death	29 (46)	18 (47)	11 (45)	>0.05

Pre-HCT Neutropenia and Lymphopenia: Associations Wuth Post-Transplant Outcomes

Pre-HCT cytopenia	Outcome	Incidence (Cytopenia)	Incidence (No Cytopenia)	p-value
Neutropenia	CsCMVI	60.0%	63.8%	0.79
	Relapse	60.0%	38.3%	0.14
	aGvHD	**93.3%**	**48.9%**	**0.002**
	Death	53.3%	44.7%	0.56
Lymphopenia	CsCMVI	62.2%	64.0%	0.88
	aGvHD	59.5%	60.0%	0.97
	Death	40.5%	56.0%	0.23

Adapted from C. V. Gomes, EBMT 2026, Poster A258

THE INTERPLAY BETWEEN CYTOMEGALOVIRUS REACTIVATION, CONDITIONING REGIMENS, AND GRAFT-VERSUS-HOST DISEASE IN ALLOGENEIC HSCT: A 10-YEAR RETROSPECTIVE ANALYSIS FROM RESOURCE-LIMITED SETTING

Najia Gul, Poster Presenter Bushra Ahsan, *AFBMTC Rawalpindi, Pakistan*
EBMT 2026, Poster A377

Acute and chronic graft-versus-host disease (aGVHD, cGVHD) remain leading complications after aHSCT in low- and middle-income countries. This single-center, 10-year retrospective cohort of 238 pediatric and adult patients evaluated the associations between CMV reactivation, conditioning regimens, and occurrence/severity of aGVHD and cGVHD, and their impact on overall survival in allogeneic HSCT recipients.

GVHD was graded by Glucksberg-Seattle criteria. Logistic regression assessed associations of aGVHD/cGVHD with CMV reactivation and conditioning regimen. Kaplan–Meier and log-rank tests evaluated OS relationships with GVHD, CMV reactivation, and conditioning intensity.

The median age of participants was 9 years (IQR 4.5–20).Donors were mainly siblings (88.7%). Conditioning regimen were:

myeloablative (MAC)	56.7%
non-MAC	29.8%
reduced intensity (RIC)	13.4%

aGVHD occurred in 71/238 (29.8%); gut aGVHD in 21.8%, liver in 7.6%, eye in 13.4%. cGVHD developed in 106/238 (44.5%), mostly limited (39.5%). Overall survival was 79.4% with mean survival 8 years post-HSCT (95% CI 7.5–8.5).

Significant associations: higher conditioning intensity (RIC) was associated with increased aGVHD (OR 4.0, p=0.03). CMV reactivation occurred in 93/238 (39%) and was associated with reduced risk of aGVHD (OR 0.26, p=0.001). On multivariate analysis, RIC remained associated with a higher incidence of aGVHD (OR 4.0, p=0.03). GVHD stage and grade significantly affected OS (p-values reported: stages p=0.003; grades p=0.02).

Real World Evidence: Special populations

COMPARATIVE ANALYSIS OF CYTOMEGALOVIRUS REACTIVATION AFTER HAPLOIDENTICAL VS MATCHED SIBLING DONOR ALLOGENEIC HEMATOPOIETIC STEM CELL TRANSPLANTATION

Marco Alejandro Jiménez-Ochoa, *Centro Médico Nacional Siglo XXI. Instituto Mexicano del Seguro Social, Mexico City, Mexico*
EBMT 2026, Poster B130

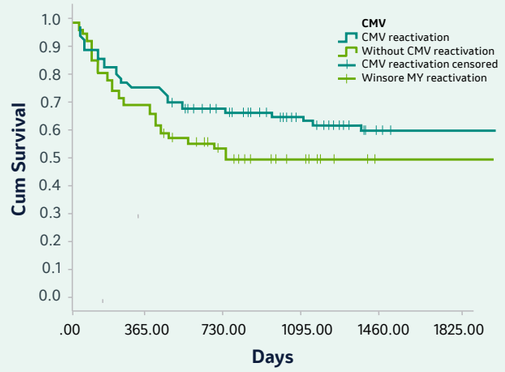
Haploidentical (haplo) grafts may induce more intense immune reconstitution and higher viral reactivation than matched related donor (MRD) grafts, but comparative data in the post-transplant cyclophosphamide (PTCy) era are limited.

This single-center, retrospective, comparative study analysed the difference in incidence, timing, clinical presentation, and outcomes (OS, DFS, and NRM) of CMV reactivation after haploidentical versus matched related donor aHSCT to identify clinically relevant differences that could guide surveillance and management. Statistical analyses was performed using SPSS v25.
The study included 182 adult allogeneic aHSCTs performed from January 2018 to December 2024: 106 MRD (58%) and 76 haplo (42%) recipients with a median age of 34 (range 17-67). Engraftment on day 12 (9-29) in the MRD group vs day 16 (13-25) in the haplo group (p=0,003). No patients received letermovir, as it was not available in Mexico until 2025. The incidence of CMV reactivation (defined as >1000 copies/mL) was significantly higher in haplo group (40/76, 53% vs 20/106, 19% in MRD; p<0,01). Both the median time to reactivation and peak CMV viral load were comparable for both groups, with no significant differences in antiviral response.

Five-year OS was comparable with 59% in the MRD group versus 53% in the haplo group. ; p>0.05). However, five-year overall survival was significantly lower among patients who experienced CMV reactivation versus those who did not (50% vs 60%; p=0.01). Leading cause of death: relapse (49%), followed by infections (23%).

The authors concluded that haploidentical aHSCT with PTCy was associated with a markedly higher incidence of CMV reactivation than matched related donor aHSCT. CMV reactivation was associated with inferior long-term survival overall, indicating clinical relevance beyond donor type.

Overall survival according to CMV reactivation status



Adapted from
M. A. Jiménez-Ochoa,
EBMT 2026,
Poster B130

Real World Evidence: Late CMV infection

LATE CYTOMEGALO-VIRUS REACTIVATION AFTER ALLOGENEIC HSCT PERSISTS IN THE ERA OF LETERMOVIR PROPHYLAXIS – A SINGLE-CENTRE REAL WORLD STUDY

Louis Peters, Poster Presenter Kirsty Sharplin, *Oxford University Hospital, Oxford, UK*
EBMT 2026, Poster B141

Patients undergoing aHSCT have impaired virus-specific immunity. Untreated CMV reactivation can cause clinically significant disease, such as colitis, pneumonitis, retinitis. Since 2019, letermovir has been funded for CMV prophylaxis during the first 100 days after aHSCT in the UK.
This single-center retrospective study (2016–2024) assessed the association between letermovir use and clinically significant CMV infection.

Among 480 aHSCT recipients, 192 (40%) were treated before letermovir introduction and 288 (60%) after. Most patients (413/480, 86%) received reduced-intensity conditioning; 397/480 were T-cell depleted (Campath 226/480, 47.1%; ATG 93/480, 19.4%; PTCy 79/480, 16.5%).
In the pre-letermovir cohort, 98/192 (51.0%) were CMV seropositive (R+), and 37/98 (37.8%) developed csCMVi. In the post-letermovir group, 140/288 (48.6%) were R+; all received letermovir prophylaxis, and 27/140 (19.3%) developed csCMVi. Among negative recipients, 4/36 (11.1%) reactivated. In a multivariate competing risk model to test whether T-cell depletion increased the risk of CMV reactivation (treating death without reactivation as a competing event), T depletion increased the risk of csCMV (HR 2.64, 95%CI 1.05-6.6, p=0.03). This remained significant after adjusting for age, sex, diagnosis and treatment era.

The median time to first reactivation pre-letermovir was 40 days (range 13-525), 37/98 (37.8%) at-risk patients reactivated by D100 post-transplant. In comparison, post-letermovir, the median time to first reactivation was 155 days (range 26-778) and 2/140 had reactivated by D100 (1.4%) (p<0.01). The median peak viral load did not significantly differ between groups.

Treatment requirements and duration for csCMVi were similar, the majority of patients receiving valganciclovir Median treatment duration was 26 days pre-letermovir (range 7–186) and 30 days in the post-letermovir cohort (range 12–54) (p=0.21). Many patients with csCMVi had concurrent GVHD and immunosuppression (pre: 18/37, 48.6%; post: 15/27, 55.6%).

The authors concluded CMV reactivation remains a frequent issue post-aHSCT. Letermovir deferred the median onset of csCMV to post-D100, but did not reduce the need for further anti-viral treatment.

LATE CYTOMEGALO-VIRUS INFECTION IN ALLOGENEIC HEMATO-POIETIC CELL TRANS-PLANT RECIPIENTS AFTER COMPLETING LETERMOVIR PRIMARY PROPHYLAXIS – A SINGLE-CENTER PRELI-MINARY ANALYSIS

Agnieszka Tomaszewska, *Medical University of Warsaw, Warsaw, Poland*
EBMT 2026, P174

Letermovir prophylaxis against CMV infection during the first 100 days after aHSCT became standard of care in seropositive recipients. Currently, published data regarding late CMV infection after completing letermovir prophylaxis are non-exhaustive and inconclusive.

In this single-center preliminary analysis, the frequency and main risk factors of any late CMV infection (CMV DNAemia and clinically significant infection-csCMVi) in 81 consecutive aHSCT patients were assessed (2022-2025); 76 patients received letermovir prophylaxis until day +100. Donors were matched sibling (n=12), matched unrelated (n=56), mismatched unrelated (n=7) haploidentical (n=6). Myeloablative conditioning was used in 59 (72%) procedures and T cell depletion in 73/81 (90%).

Among the key findings were:

- In this cohort 35 (43%) late CMV infections occurred, though most cases demonstrated low viral load and were self-limiting. Only 6 (6/35, 17%; 6/81, 7% overall) infections were csCMVi and required antiviral therapy
- Among 29 CMV-seronegative donors, late CMV infection developed in 15 recipients, with 4 csCMVi
- Ten patients had CMV “blips” during letermovir; 5 later developed late CMV infection and only in 1 it was classified as clinically significant
- Co-infections were common: 17/35 (47%) with CMV had other viral pathogens (EBV in 12, BKV in 3, HHV 6 in 1 and AdV in 1 patient respectively)
- During follow-up, 11 recipients developed acute GVHD and 17 chronic GVHD requiring additional immunosuppressive treatment; 13 received ruxolitinib, of whom 10 (77%) had late CMV infection but only one had csCMVi
- Relapse occurred in 15/76 (20%); CMV DNA was detected in 8 (53%) of these. Thirteen patients (17%) died; none of the deaths were directly attributed to late CMV, though 6 of the deceased presented a late infection at observation

In conclusion, letermovir markedly reduced csCMVi, but late CMV infection occurred in 43% of recipients after prophylaxis ended — mostly low level and self limiting. Larger studies are needed to identify which patients might benefit from extended letermovir prophylaxis.

2. Co-infection: EBV co-infection

EBV REACTIVATION AND PTLD IN THE LETERMOVIR ERA: BEYOND ANTIVIRAL PROPHYLAXIS

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EBMT 2026, Poster B086

Reactivation of Epstein–Barr virus and post-transplant lymphoproliferative disorders (PTLD) after aHSCT, occurs in 1–3% of unselected recipients. In high-risk settings, including in vivo T-cell depletion, specific donor types, and severe aplastic anemia incidence increases to 8–10%. Emerging evidence speculates a possible rise in EBV-R and PTLD in the letermovir era. This retrospective analysis (2019-2025) assessed 196 aHSCT patients with minimum 6 months' follow-up on EBV-R and PTLD incidence. Clinical and transplant variables were compared annually, including EBV/CMV serostatus, conditioning intensity, GVHD prophylaxis, letermovir use, GVHD, corticosteroid exposure, infections, relapse, and immune reconstitution at day +30.

EBV-R occurred in 30/196 (15.3%) and PTLD in 14/196 (7.1%); rates rose over time and peaked in 2023 (EBV-R 22.7%, PTLD 18.2%). Baseline transplant characteristics and transplant-related complications did not differ between EBV-R and PTLD groups, except for immune reconstitution parameters, including NK-cells ($p=0.0019$), activated NK/T-cells ($p=0.0266$), and the CD4/CD8 ratio ($p=0.0266$).Univariate analysis linked EBV-R with ATG exposure ($p<0.001$), PTCy use ($p<0.001$), higher Fujimoto risk score ($p=0.0056$), with a trend for letermovir exposure ($p=0.085$), and differed by transplant type ($p<0.001$). PTLD was significantly associated with EBV reactivation ($r=0.65, p<0.001$), ATG use ($r=0.24, p=0.0006$), PTCy ($r=-0.21, p=0.0028$), transplant type ($r=-0.16, p=0.023$), and Fujimoto risk ($r=0.14, p=0.047$), but not with CMV reactivation.

The median onset of EBV-R/PTLD was 42 days (IQR 38.3–54.5), therefore day 30 immune profiles were examined. Patients who later developed EBV R/PTLD had higher white blood cell counts, with increased NK and B-cells, but lower CD3+, CD4+, CD8+, and activated NK/T cells (all $p \leq 0.002$). ROC analyses identified NK, WBC, and B cells as the best predictors for EBV-R. Multivariable models showed higher day 30 CD4+ (OR 0.82, $p=0.05$) and activated NK/T cells (OR 0.71, $p<0.01$) were independent protective factors, while higher B cells increased risk for EBV-R and PTLD ($p=0.05$).

Based on these RWE data, EBV-R and PTLD increased over time and were associated with ATG, PTCy, and donor type. Day-30 immune reconstitution was a key risk factor. The key take-away based on this RWE data, suggests that targeted early immune monitoring could improve risk stratification.

CMV AND EBV REACTIVATION AFTER HAPLOIDENTICAL HEMATOPOIETIC CELL TRANSPLANTATION IN ELDERLY PATIENTS WITH HEMATOLOGICAL MALIGNANCIES

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EBMT 2026, Poster B113

Elderly patients (≥60 years) undergoing haploidentical HCT face an increased risk of CMV and EBV reactivation following immunosuppressive regimens. However, data regarding viral management and its prognostic impact specifically in elderly recipients remains limited. This multicentre retrospective study of 74 first-time haplo-HCT recipients examined CMV and EBV reactivation, early risk factors, and effects on transplant outcomes. The primary endpoint was overall survival; secondary endpoints included the incidence of CMV/EBV reactivation, GVHD, PFS, cumulative incidence of relapse (CIR), and NRM.

Among the key findings were:

- GVHD prophylaxis: ATG in 83.8% (n=63); 17.6% received PTCy (n=13). Pre-transplant antivirals: ganciclovir 89.2% (n=66); foscarnet 21.6% (n=17). Post-transplant prophylaxis: letermovir in 18.9% (n-14)
- Median time to reactivation: CMV 1.7 months, EBV 2.7 months. CMV peaked within 3 months
- Cumulative incidences: CMV 45.9% at 3 months (95% CI, 34.2%-56.9%) and 52.8% at 6 months (95% CI, 40.7%-63.5%); EBV 15.0% at 3 months (95% CI, 7.9%-24.0%) and 21.7% at 6 months (95% CI, 13.1%-31.8%) respectively. CMV–EBV co-reactivation: 10.8% by 3 months (95% CI, 5.0%-19.1%) and 12.2% by 6 months (95% CI, 6.0%-20.8%)
- Letermovir was identified as independently protective factor for CMV reactivation (HR 0.37, P=0.039) but was independently associated with higher EBV reactivation (HR 3.14, P=0.015)
- CMV reactivation within ≤100 days did not affect OS, PFS, or NRM but was linked to higher relapse risk (CIR HR 5.50, P=0.031)
- EBV reactivation within ≤180 days was associated with worse OS (HR 3.51, P=0.006) and PFS (HR 2.60, P=0.033) but not with CIR or NRM

The authors concluded that in elderly haplo-HSCT recipients, EBV reactivation correlated with inferior survival, while early CMV reactivation predicted disease relapse without compromising survival. Letermovir lowered CMV risk but may increase EBV reactivation, underscoring the need for balanced prophylaxis and close viral monitoring in these vulnerable patients.

Co-infection: co-infection in pediatric patients

POST-TRANSPLANT LYMPHOPROLIFERATIVE DISEASE IN PEDIATRIC PATIENTS RECEIVING LETERMOVIR AS CMV INFECTION PROPHYLAXIS

Katarzyna Smalisz, *University Children's HospitalInstitute, Cracow, Poland*
EBMT 2026, Poster B221

After allogeneic HSCT, EBV reactivation can cause post-transplant lymphoproliferative disorder (PTLD). Letermovir used for CMV prophylaxis has been associated with increased EBV reactivation.

The goal of this retrospective, single center study was to assess the occurrence and severity of EBV reactivation/PTLD in children receiving letermovir for CMV prophylaxis after HSCT.

The analysis included 72 allo-HSCT procedures between 2023 and 2025, letermovir prophylaxis was given to 34 children. The analysis focused on patients with EBV reactivation requiring preemptive therapy.

EBV reactivation occurred in 18% (6/34) among children on letermovir. Time to reactivation ranged from 39 to 123 days post transplant, with a median 79 days. In one patient the infection was asymptomatic; 5 (83%) developed PTLD (painful lymphadenopathy, fever, pancytopenia). All 6 patients were treated with rituximab; 2 progressed to non-Hodgkin lymphoma. No PTLD-related deaths in the cohort.

Intensive EBV monitoring, especially around 3 months post-HSCT and in children >14 years, is recommended.

CMV AND BK VIRUS REACTIVATION AFTER ALLOGENEIC HSCT ACCORDING TO DONOR TYPE: A SINGLE-CENTER PEDIATRIC EXPERIENCE

Cristina Jercan, *Carol Davila University of Medicine and Pharmacy/Fundeni Clinical Institute Bucharest, Romania*
EBMT 2026, Poster B259

CMV and BKV are among the most frequent viral complications in pediatric aHSCT recipients. This single-center retrospective cohort study examined the incidence of CMV - and BKV reactivation rates across donor types, timing to reactivation, viral load, clinical severity, need for therapy. Assessment of the impact of letermovir prophylaxis was a secondary endpoint. CMV reactivation was defined by DNAemia requiring pre-emptive therapy; BKV reactivation defined by viruria/viremia and clinical/diagnostic features.
Patients (n= 191) were included from January 2013 until November 2023. Evaluated donor groups were matched sibling donor (MSD), matched unrelated donor (MUD), and haploidentical donor (Haplo).

CMV reactivation occurred in 43,5% (83/191, p=0.040) overall. Rates by donor type: MSD 26.2%, MUD 47.6%, Haplo 50.0% (p=0.037 vs MSD). D-/R+ mismatch was the strongest predictor for reactivation (59,4%; overall p=0.040). Letermovir did not significantly reduce CMV reactivation incidence across donor group, due to the small subgroup sizes and use in the highest-risk patients, however, in the letermovir group zero CMV-related complications occurred, and this group demonstrated 43% lower peak viremia compared to non-recipients.
BKV reactivation was 53,4% and was comparable for all donor types, 18,6% of BKV-positive patients developed haemorrhagic cystitis, regardless of donor source.

The authors concluded that universal surveillance and risk-adapted prophylaxis (including letermovir for high-risk patients) plus standardized BKV monitoring are recommended for pediatric post-HSCT viral management.

3. Refractory/resistant CMV infection: Treatment refractory/resistant infections

REAL-WORLD USE, EFFECTIVENESS AND SAFETY OF MARIBAVIR IN HEMATOPOIETIC CELL TRANSPLANT RECIPIENTS WITH REFRACTORY/RESISTANT CMV OR INTOLERANCE TO ANTI-CMV AGENTS: INTERIM ARISE RESULTS

Johan Maertens, *UZ Leuven Leuven, Belgium*
EBMT 2026, Poster B073

Maribavir was approved in Europe in 2022 for treatment of post-transplant refractory/resistant (R/R) cytomegalovirus (CMV) infection/disease. Real-world evidence was needed to describe its use, effectiveness and safety in hematopoietic cell transplant (HSCT) recipients. The poster reported interim ARISE study data for allogeneic HCT patients.

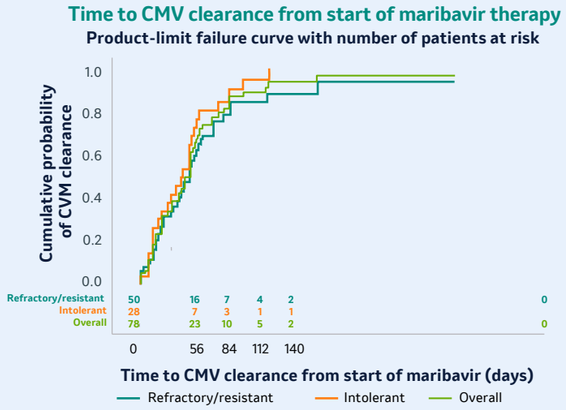
ARISE is a multinational, retrospective cohort study of adult (≥18 y) HCT or solid-organ transplant recipients with R/R CMV or intolerance to anti-CMV agents. The primary objective was to describe CMV clearance at maribavir discontinuation (last negative CMV quantitative PCR before stopping maribavir). The interim analysis included 89 allogeneic HCT recipients (56 with R/R CMV; 33 intolerant to anti-CMV agents) from nine European countries.

Investigators retrospectively collected demographic, clinical, treatment pattern, safety and healthcare resource utilization (HCRU) data. Outcomes were summarized with descriptive statistics and Kaplan–Meier analysis. Follow-up median was 14.6 months (Q1–Q3: 9.1–21.2).

Among 74 evaluable patients at maribavir discontinuation, CMV clearance was observed in 38/47 (80.9%; 95% CI: 66.7–90.9) of R/R recipients and 25/27 (92.6%; 95% CI: 75.7–99.1) of intolerant recipients. Median time to clearance from maribavir start was 50 days (Q1–Q3: 26–71) in the R/R group and 44 days (18–56) in the intolerant group. Median maribavir course duration (n=94 courses) was 49.5 days (22–61); 53.1% of courses were initiated at home (51/96). After initiation of maribavir, myelosuppression incidence rates per person-year fell from 0.623 to 0.181 (R/R) and from 1.409 to 0.218 (intolerant); nephrotoxicity rates fell from 0.147 to 0.052 (R/R) and from 0.634 to 0.054 (intolerant). Hospitalization rates per person-year decreased after maribavir: from 1.36 to 0.91 (R/R) and 1.27 to 0.46 (intolerant). All-cause mortality was 41.1% (R/R) and 42.4% (intolerant).

The investigators concluded that interim real-world data supported maribavir's effectiveness and safety profile in HCT recipients with R/R CMV or intolerance to anti-CMV agents. CMV clearance rates at discontinuation (80.9% R/R; 92.6% intolerant) appeared higher than previously observed in the SOLSTICE trial, and myelosuppression and nephrotoxicity rates declined after maribavir initiation, consistent with its lower hematologic and renal toxicity versus historical antivirals.

Adapted from J. Maertens, EBMT 2026, Poster B073



VIROLOGIC OUTCOMES BY BASELINE CMV VIRAL LOAD IN HCT RECIPIENTS TREATED WITH MARIBAVIR: POST HOC ANALYSIS OF PHASE 2 AND 3 TRIALS

Genovefa A. Papanicolaou, *Memorial Sloan Kettering Cancer Center, New York, United States of America (the)*
EBMT 2026, Poster B080

Higher baseline CMV DNAemia (viral load) has been associated with delayed DNAemia clearance in addition to host factors. This exploratory, post hoc analysis of two maribavir trials in HCT recipients with refractory CMV infection examined virologic and safety outcomes by baseline DNAemia in HCT recipients with refractory CMV infection treated with maribavir in clinical trials.

Patients received maribavir for up to 24 weeks in the phase 2 dose-ranging study (SHP620-202) and 8 weeks in the phase 3 SOLSTICE trial. Analyses were restricted to maribavir-treated patients. Baseline plasma CMV DNAemia was categorized as low/intermediate or high (study 202: ≥10,000 copies/mL, SOLSTICE: ≥91,000 IU/mL). The primary endpoint was DNAemia clearance by week 8 and at any time on study. Safety endpoints included the incidence of adverse events (AEs), treatment-emergent AEs (TEAEs), and AEs leading to treatment interruption or discontinuation, or death.

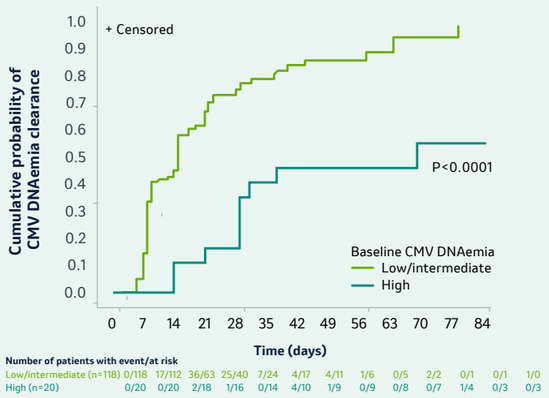
A total of 138 aHSCT patients treated with maribavir were included, 118 (85.5%) with low/intermediate and 20 (14.5%) with high baseline plasma CMV DNAemia. Median age was 55 years (ranging from 19-77) in the low/intermediate group and 60.5 years (ranging from 26-69) years in the high DNAemia groups.

By week 8, 64.4% (76/118) of patients with low/intermediate baseline DNAemia achieved clearance versus 40.0% (8/20) in the high baseline DNAemia group. DNAemia clearance at any time was achieved in 82.2% (97/118) of the low/intermediate DNAemia group and 45.0% (9/20) of the high DNAemia group. Time to first DNAemia clearance was significantly shorter ($p<0.0001$) for patients with low/intermediate versus high baseline DNAemia (at any time up to week 12).

Overall AE incidence was similar between DNAemia groups. however, AEs leading to treatment interruption or discontinuation, or death, occurred more frequently among patients with high versus low/intermediate baseline DNAemia.

These data reflect a population with complex treatment needs, especially in those with high DNAemia, and further research is warranted to determine if early identification and initiation/switch to maribavir could improve clinical outcomes in patients with refractory post-HCT CMV.

Kaplan-Meier analysis for time to CMV DNAemia clearance



Adapted from G. A. Papanicolaou, EBMT 2026, Poster B080

EFFICACY AND SAFETY OF MARIBAVIR IN HEMATOPOIETIC CELL TRANSPLANT RECIPIENTS BY BASELINE NEUTROPENIA STATUS: A POST HOC ANALYSIS OF PHASE 2 AND 3 CLINICAL TRIALS

Alicja Sadowska-Klasa, *Medical University of Gdansk, Gdansk, Poland*
EBMT 2026, Poster B090

Maribavir has demonstrated efficacy in refractory CMV infection and is associated with less myelotoxicity compared with ganciclovir/valganciclovir; however, data in neutropenic HCT recipients remain limited. This post hoc analysis of clinical trial data assessed the efficacy and safety of maribavir according to baseline neutropenia status.

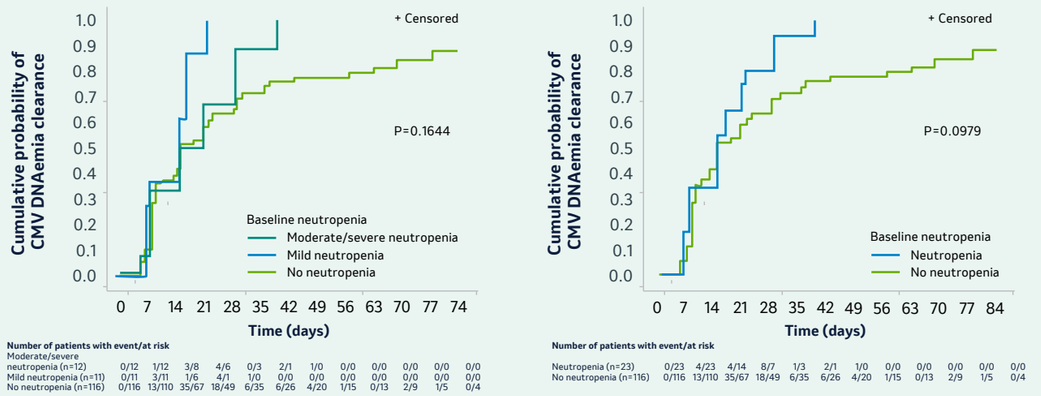
In total, 139 maribavir-treated patients from the phase 3 SOLSTICE trial and a phase 2 dose-ranging study were included. Patients were stratified by baseline neutrophil count, with 9% (n=12) having moderate-to-severe neutropenia, 8% (n=11) mild neutropenia, and 83% (n=116) no neutropenia.

At week 8, CMV DNAemia clearance was achieved in 83.3% (n=10/12) of patients with moderate-to-severe neutropenia and 63.6% (n=7/11) with mild neutropenia, compared with 58.6% (n=68/116) in patients without neutropenia. Clearance at any time during the study was observed in 91.7% (n=11/12), 81.8% (n=9/11), and 75.0% (n=87/116), respectively. Time to CMV clearance, as assessed by Kaplan–Meier analysis, was similar across groups ($p=0.1644$), as well as between those with and without neutropenia ($p=0.0979$).

Treatment-emergent adverse events (TAEs) were reported in 83.1% of the moderate-to-severe neutropenia group, 74.5% in the mild neutropenia group, and 68.4% in the group without neutropenia, with comparable severity across groups. Discontinuation due to adverse events occurred in 25.0% (n=3/12), 9.1% (n=1/11), and 28.4% (n=33/116), respectively.

These findings suggest that maribavir maintains efficacy and a consistent safety profile irrespective of baseline neutropenia, although results should be interpreted with caution given the small subgroup sizes.

Adapted from A. Sadowska-Klasa, EBMT 2026, Poster B090



MARIBAVIR FOR TREATMENT OF REFRACTORY/ RECURRENT CYTOMEGALOVIRUS INFECTION IN ALLOGENEIC HSCT RECIPIENTS

Aleksandr Siniaev, *RM Gorbacheva Research Institute, Pavlov University, Saint Petersburg, Russia*
EBMT 2026, Poster B095

This single-center study evaluated virologic response, durability, safety signals, and six-month outcomes of maribavir in aHSCT recipients with refractory or recurrent CMV (R/R) in clinical practice.

The study included 50 aHSCT recipients with median age 40 (43/50, 86% adults) with refractory (41/50, 82%) or recurrent (9/50, 18%) CMV. The median time from aHSCT to CMVi was 36,5 days (range -1 - 342), after which 86% (43/50) received PET with ganciclovir for median 14 days (range 2-75). Three patients were treated for CMV pneumonia and three for GI disease.

Maribavir was used for R/R patients in 36 patients (72%) and later line in another 14 patients (28%). Median duration from CMVi onset until the start of maribavir was 18,5 (9-486) days; median treatment was 25,5 (2-75) days. No CMV resistance testing was performed.

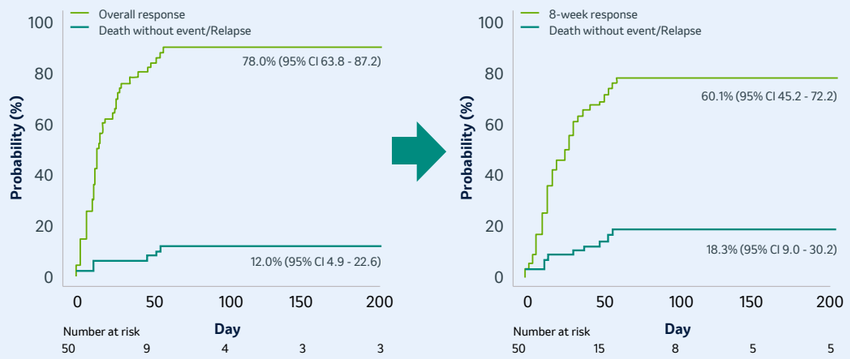
At six months following results were found:
- Cumulative incidence of recurrent CMV 38.1% (95% CI 23,9–52,3)
- Overall therapy failure 47,4% (95% CI 32,4-61,1)
- Overall survival 71.9% (95% CI 56,4–82,8)
- Failure-free survival 35,9% (95% CI 22,2–49,8).

The overall cumulative incidence (CI) of maribavir response at 6 months was 78.0% (95% CI 63.8–87.2), and at 8 weeks it was 60,1% (95% CI 45,2-72,2)

Grade 3–4 cytopenias at 4 weeks were common (thrombocytopenia 54.9%; neutropenia, leukopenia, anemia, each ~31%), but causal attribution to maribavir was not established. Reactivation of other herpesviruses occurred frequently (HHV-6 62.7%; EBV 23.5%; HSV 19.6%; zoster 3.9%).

Conclusion: In this real-world aHSCT cohort, maribavir achieved rapid CMV clearance in the majority of patients, similar to clinical trials, but durable control was limited with substantial recurrent CMV and frequent cytopenias and other herpesvirus reactivations. Close virologic monitoring and management of concurrent herpesvirus infections are essential during maribavir therapy.

6-month maribavir therapy overall response and 8-week response



Adapted from A. Siniaev, EBMT 2026, Poster B095

PHYSICIAN SURVEY OF REAL-WORLD MARIBAVIR USE AND TREATMENT DECISION MAKING IN REFRACTORY CYTOMEGALOVIRUS INFECTION AFTER TRANSPLANTATION

Johan Maertens, *UZ Leuven, Leuven, Belgium*
EBMT 2026, Poster B129

Management of refractory or resistant cytomegalovirus (CMV) after allogeneic hematopoietic stem cell transplantation (aHSCT) lacks standardized definitions and treatment approaches. Heterogeneity in clinical practice may affect outcomes and choice of antivirals such as maribavir. The goal of this cross-sectional, web-based physician survey, part of the ARISE multinational, non-interventional, retrospective cohort study across Europe that includes a cross-sectional survey of physicians from 43 participating sites, was to describe real-world physician practices, definitions, decision drivers, and treatment choices for refractory/resistant CMV after aHSCT across multiple countries.

Most respondents (n=39) were transplant/ infectious disease physicians (33,3% hematologists, 33,3% nephrologists, 20,5% infectious diseases specialists) from academic centers with varying annual aHSCT and CMV caseloads from 10 different countries.

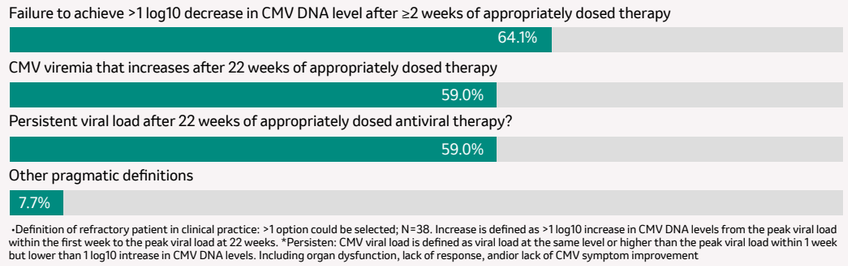
75% reported following institutional and/or national guidelines, but practice variation persisted. There was wide variation in what physicians considered refractory or resistant. Especially tresholds and timeframes varied.

Almost half of respondents perceived a gap between the guidelines/literature definition of refractory CMV and clinical practice, or were unsure about definition versus clinical practice (resp 20.5% and 28.5%). Influences of the availability of new therapies on treatment approaches were faster switches to other treatment options when chosen treatment does not seem to be effective (71.8%) or at first sign of treatment related toxicities (56.4%), and the use of treatment options with a lower expected toxicities (71.8%). Viral load levels (dynamics 32.6% absolute levels 15.8%) and donor/recipient CMV serostatus (15.8%) were the most important characteristics for decision-making.

Maribavir was discontinued when the viral load was undetectable in 66.7%, where continuation after CMV viremia clearance was done to avoid recurrent infection in 65.8% (n=38). 73.7% of the respondents (n=38) continued maribavir treatment beyond 8 weeks in case of a persistent viral load. Discontinuation of dose reduction of anti-CMV treatment was mainly due to treatment-limiting toxicities.

Authors concluded that in general international guidelines are followed, although there is also a perceived gap about definitions versus clinical practice. A reason may be that the 4th international Guidelines may not have been available at the time of completing the survey.

Responses for definitions of refractory CMV



•Definition of refractory patient in clinical practice: >1 option could be selected; N=38. Increase is defined as >1 log10 increase in CMV DNA levels from the peak viral load within the first week to the peak viral load at 22 weeks. *Persisten: CMV viral load is defined as viral load at the same level or higher than the peak viral load within 1 week but lower than 1 log10 intrease in CMV DNA levels. Including organ dysfunction, lack of response, andior lack of CMV symptom improvement

Adapted from J. Maertens, EBMT 2026, Poster B129

RISK FACTORS FOR RECURRENT CYTOMEGALOVIRUS INFECTION IN HEMATOPOIETIC STEM CELL AND SOLID ORGAN TRANSPLANT RECIPIENTS: A SCOPING REVIEW

Wenjian Mo, *Poster Presenter Shunqing Wang, Guangzhou First People's Hospital Guangzhou, China* EBMT 2026, Poster P088

Recurrent CMV infection is common after allogeneic HSCT and solid-organ transplantation. Beyond raising the risk of end-organ disease and graft rejection, recurrent CMV can delay immune recovery, increase susceptibility to other opportunistic infections, and raise mortality. Early identification of high risk patients is therefore key to preventing complications and improving long term outcomes. This PRISMA-ScR scoping review (2010-2023) aimed to identify risk factors associated with recurrent CMV infection in HSCT and SOT recipients. The researchers performed a systematic search for literature on risk factors for refractory CMV infection after transplantation was conducted in eight databases including PubMed, EMBASE, the Cochrane Library, Web of Science, CNKI, Wanfang, VIP and CBM.

Ten studies met the inclusion criteria; 5 studies in aHSCT (n=331) and 5 studies in SOT (n=217). Across these studies, a total of 21 risk factors for recurrent infection were identified, 9 specific to HSCT and 12 for SOT. A summary of the findings you find in the table below.

This review showcases host-, transplant-, and virus related risk factors for CMV recurrence in both HSCT and SOT populations. Early identification of high risk patients can guide optimized antiviral strategies (including drug choice and duration) to reduce recurrence, limit complications, and improve outcomes.

Hot map of risk factors for recurrent CMV infection.

Transplant type	Factor category	Sub-category	Factors	Nr of recurrent reference
Haematology transplant (n = 5)	**Host-related factors**	Rejection	acute or chronic GVHD, any grade (yes vs. no)	2
			aGVHD (grade II-IV vs. grade 0-II)	2
		Primary Disease	Diagnosis AML/MDS (yes vs. no)	1
		Age	Higher recipient age	1
		Response to CMV treatment	Response to treatment (refractory vs. not refractory)	1
		CMV-specific immune response	CMV-specific CD8+ TcM at day 30 (µL) (<0.032 vs. ≥0.032)	1
	Transplantation-related factors	Serological status	CMV serostatus (D+R+ vs. R-)	1
			CMV serostatus (D+/R- vs. R-)	1
		Donor type or source	Donor type (cord blood vs. non-cord blood)	1
			Donor type (HBMT vs. MSD)	1
			Donor type (mismatched family vs. MSD)	1
			Donor type (mismatched vs. matched)	2
		Immunosuppressive-related drugs	Treatment with high-dose steroids after day 100 (>1 mg/kg) (with vs. without)	1
Organ transplant (n = 5)	**Host-related factors**	Primary Disease	CKD (yes vs. no)	1
		Genotyping	NFKB1 genotype (ins/ins vs. ins/del-del/del)	1
		Transplantation-related complications	Pneumonia (yes vs. no)	1
		Rejection reaction	Rejection within 3 mo prior (yes vs. no)	1
	Transplantation-related factors	Serological status	CMV serostatus (D+/R- vs. others)	1
		Patient recovery following transplantation	Delayed graft function (days)*	1
		Transplantation types	Transplant type (lung vs. non-lung)	1
	Viral-related factors	CMV mRNA	Baseline CMV mRNA detection-UL22A-5p (yes vs. no)	1
		CMV DNA load	CMV load half-life, days (>3 vs. <3)	1

Notes: The numbers in the hot map indicate the number of articles corresponding to the factors.

Adapted from Wenjian Mo, EBMT 2026, Poster P088

RISK FACTORS FOR REFRACTORY CYTOMEGALOVIRUS INFECTION IN HEMATOPOIETIC STEM CELL AND SOLID ORGAN TRANSPLANT RECIPIENTS

Wenjian Mo, **Poster Presenter Shunqing Wang, Guangzhou** *First People's Hospital Guangzhou, China*
EBMT 2026, Poster A217

Refractory cytomegalovirus (CMV), defined as failure to achieve ≥1 log10 viral load reduction after 2 weeks of antiviral therapy or presence of resistant CMV with genotypic mutations, has a negative impact on outcomes after aHSCT and solid organ transplant (SOT). A scoping review framework (PRISMA-ScR) was employed to systematically search literature for risk factors for refractory/refractory-resistant CMV, which can guide monitoring and early management.

Thirteen studies were included (10 HSCT, 3 SOT) comprising 946 HSCT and 101 SOT patients. Evidence was based on the number of studies reporting each factor.

These factors, supported by multiple cohort studies, can inform intensified monitoring, earlier resistance testing, and consideration of alternative therapies to improve viral clearance and patient outcomes.

Hot map of risk factors for recurrent CMV infection

Transplant type	Factor category	Factors	Nr. of refractory reference	Nr. of resistance reference
Haematology transplant (n=10)	**Host-related factors**	aGVHD (grade II-IV vs. grade 0-I)	4	
		aGVHD (yes vs. no)	2	
		EB viremia (yes vs. no)	1	
		NKG2C (delidel vs. wt/del)	1	
		NKG2C (delidel vs. wt/wt)	1	
		Platelet count at day 90 after transplant ($\times 10^9$/L) (≤86 vs. >86)	1	
	Transplantation-related factors	ATG used in conditioning therapy (yes vs. no)	1	
		CMV serostatus (D-/R+ vs. D+/R+)	1	
		Conditioning regimen (TBI/Cy vs. Bu/Cy)	1	
		Donor type (cord blood vs. non-cord blood)	1	
		Donor type (Haplo or MUD vs. MSD)	1	
		Donor type (HBMT vs. MSD)	1	
		Donor type (mismatched famity vs. MSD)	1	
		Prophylactic immunosuppression (MMF vs. MZR)	1	
		Stem cell source (BM or CB vs. PB or PB+BM)	1	
		Steroid use (MP, mg/kg/day) (≥1 vs. <1)	1	
		WBC at 14 day after transplant ($\times 10^9$/L) (<4 vs. ≥4)	1	
	Viral-related factors	CMV-specific CD8+ TCM at day 30 (µL) (<0.032 vs. ≥0.032)	1	
		Highest CMV viral load ($\times 10^4$ copies/mL) (>1 vs. ≤1)	2	
Organ transplant (n=3)	**Anti-CMV drug-related factors**	History of VGCV underdosing or low plasma concentration (yes vs. no)		1
		VGCV prophylaxis at infection onset (yes vs. no)		1
	Host-related factors	Persistent clinical disease (yes vs. no)		1
		Younger recipient age		1
	Transplantation-related factors	CMV serostatus (D+/R- vs. others)	1	1

Adapted from Wenjian Mo, EBMT 2026, Poster A217

Refractory/resistant infection in pediatric patients

IMPACT OF REFRACTORY PRE-TRANSPLANT CYTOMEGALOVIRUS INFECTIONS ON SURVIVAL IN CHILDREN UNDERGOING STEM CELL TRANSPLANT FOR PRIMARY IMMUNODEFICIENCY DISORDERS

Fatema Al-Hashem, *King Faisal Specialist Hospital and Research Center Riyadh, Saudi Arabia*
EBMT 2026, Poster P075

Pre-transplant CMVi has been associated with worse transplant outcomes. This retrospective, single-center analysis investigated the impact of refractory pre-transplant CMV infection on overall survival after aHSCT in pediatric patients with primary immunodeficiency disorders (PIDs).

Transplant-naïve pediatric patients with CMV refractory to antiviral therapy were included. Patients who responded to therapy were excluded. CMVi was defined as 500 copies/mL before stem cell infusion. Kaplan–Meier survival analyses were performed (SPSS v20). Number of transplants considered eligible for analysis was 318. The mediam age was 0.9 years (0.5 months–14 years), 70.8% had myeloablative conditioning, 2.2% reduced intensity. Matching results were:

- 65,1% fully matched
- 14.8% haploidentical
- 10.1% cord blood
- 7.5% matched-unrelated
- 2.5% mismatched donors

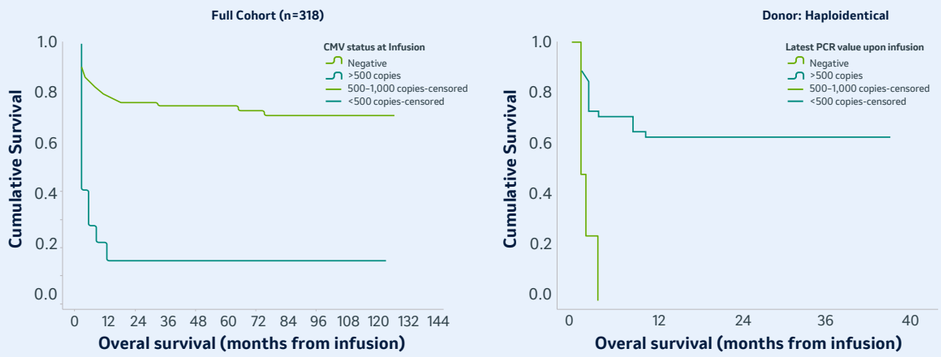
At infusion, 4.4% (14 patients) were CMV seropositive.

At a median follow-up of 38.2 months the cumulative probability of 5-year overall survival was 74.8% in seronegative recipients versis 14.3% in seropositive recipients ($p<0.001$). Especially in haploidentical recipients seronegative patients a large gap in cumulative 5-year overall survival probability ($p<0.001$) was observed. Cord blood transplants did not demonstrate statistical difference between CMV- seronegative and -seropositive patients.

Refractory pre-transplant CMV infection markedly reduced survival after allo-SCT in pediatric PID patients, particularly among haploidentical recipients. The authors concluded that adoptive immunotherapy (cytotoxic T-lymphocytes) may be considered to improve outcomes in these high-risk patients.

Adapted from F. Al-Hashem, EBMT 2026, Poster P075

Survival analysis of all 318 recipients & B) Survival analysis of haploid identical recipients (P<0.001).



4. Novel therapeutic & prophylactic approaches

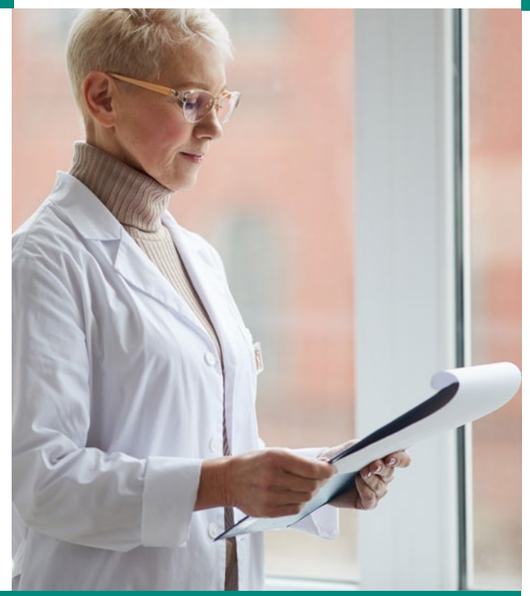
LOW-DOSE WEEKLY CIDOFOVIR AS AN EFFECTIVE PROPHYLACTIC ALTERNATIVE TO LETERMOVIR FOR PRE-ENGRAFTMENT CMV IN ALLOGENEIC HSCT

Santanu Sen, *Kokillaben Dhirubhai Ambani Hospital, Mumbai, India*
EBMT 2026, Poster B066

In many resource-limited regions, letermovir remains inaccessible and due to the myelosuppressive toxicity of ganciclovir it is limited for prophylactic use. The need for effective alternatives led to the prospective analysis of low-dose weekly cidofovir for pre-engraftment CMV prophylaxis compared to benchmark efficacy and safety profile of letermovir in high-risk aHSCT patients (R+/D-, haploidentical with PTCy, prior CMV infection). The key outcomes of this prospective study were reactivation rates, need for secondary therapy, engraftment kinetics, nephrotoxicity, and survival and were compared to published letermovir data.

Among 32 patients receiving cidofovir prophylaxis, the breakthrough CMV reactivation rate was 31% (10/32), comparable to the 35-40% range reported with letermovir. Of these 10, 70% (7/10) were controlled by escalating cidofovir to a treatment dose, one patient (3.1%) required ganciclovir. No significant nephrotoxicity or attributable mortality was observed, mirroring the safety profile of letermovir. Engraftment kinetics were similar as well, with a median neutrophil engraftment at day +14.

In summary, low-dose weekly cidofovir demonstrated comparable breakthrough rates and a safety profile as letermovir for prophylaxis. For geographical regions where letermovir is unavailable or cost-prohibitive, this could provide a potential effective, practical, and affordable alternative, without the myelosuppressive risks of ganciclovir.



PROGNOSTIC IMPLICATIONS OF CMV INFECTION/ DISEASE AND HHV-6 ENCEPHALITIS IN CORD BLOOD TRANSPLANT RECIPIENTS RECEIVING FOSCARNET

Shogo Matsui, *Toranomon hospital Tokyo, Japan* EBMT 2026, Poster B081

Beyond CMV, HHV 6 encephalitis is a frequent and severe complication after cord blood transplantation (CBT), especially when there is a high prevalence among the population. Foscarnet is active against both viruses, but large studies of prophylactic use of foscarnet in CBT are scarce.

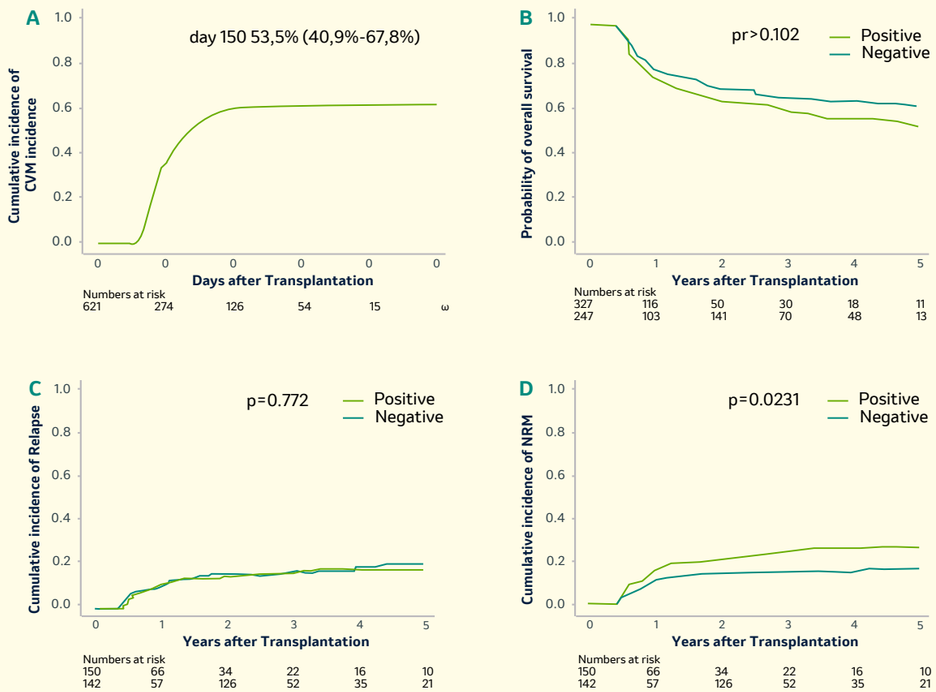
In this retrospective analysis, 491 patients underwent single unit CBT (2016–2022) and received prophylactic foscarnet from day 7 to day 50 post transplant (and no letermovir). Patients received tacrolimus plus mycophenolate mofetil (MMF) for GVHD prophylaxis. The primary endpoints were the cumulative incidence of CMV infection and its effect on relapse, non relapse mortality (NRM), and overall survival (OS); the secondary endpoints included CMV disease and HHV 6 encephalitis and impact on clinical outcomes. Landmark analyses up to day 150 evaluated the impact of these viral infecitons and aGVHD.

By day 150, CMV infection occurred in 53.5% of patients, more often in those ≥60 years (51.0% vs. 37.1%, p<0.01) and after reduced intensity conditioning (20.4% vs. 11.3%, p<0.01).. In univariate analysis CMV infection was linked to higher NRM (23.9% vs. 15.5%, p=0.023) but not to 3-year cumulative relapse or OS (15.7% vs. 16.2%, p=0.77 and 60.5% vs. 66.1%, p=0.102 respectively. Multivariate analysis found no independent effect of CMV infection on relapse, NRM, or OS. CMV disease by day 150 was rare (4.1%), though was associated with higher NRM (57.9% vs. 18.5%, p<0.01) and inferior OS (27.0% vs. 64.6%, p<0.01). See Figure.

HHV 6 encephalitis occurred in 9.2% by day 150, but did not significantly change NRM (23.4% vs. 20.4%, p=0.965) or OS (62.9% vs. 62.7%, p=0.607). Foscarnet was stopped within 30 days in 41.5% of patients, mostly for renal dysfunction (155; 75.5%); kidney function improved in in 122 patients after stopping the drug.

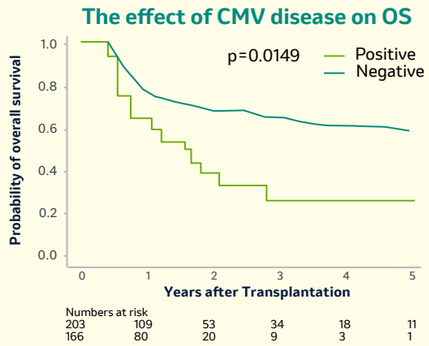
In conclusion, prophylactic foscarnet in CBT did not sufficiently prevent HHV-6 encephalitis. In the patients on foscarnet prophylaxis, CMV infections were linked to higher NRM and CMV disease adversely affected OS, Renal toxicity limited foscarnet use in many patients.

Cumulative incidence of CMV infection (A) and its effect on OS (B), the incidence of relapse (C) and NRM (D)

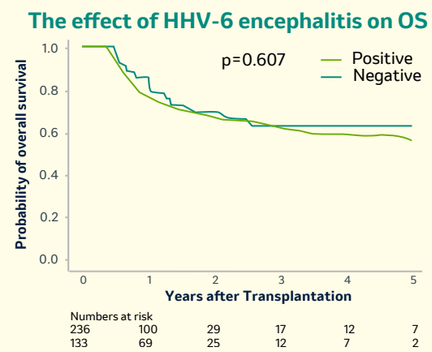


Adapted from Shogo Matsui, EBMT 2026, Poster B081

The effect of CMV disease on OS



The effect of HHV-6 encephalitis on OS



MANAGEMENT OF CMV INFECTION WITH MARIBAVIR IN PATIENTS TREATED WITH CAR-T THERAPY

Cristian Gutierrez-Padilla, *University Hospital 12 de Octubre, Madrid, Spain* EBMT 2026, Poster B127

CMV reactivation after CAR-T therapy poses a therapeutic challenge due to the combined toxicities of both CAR-T therapy and conventional antivirals. This single-centre retrospective study evaluated the efficacy and safety of maribavir for pre-emptive treatment of CMV reactivation in patients treated between September 2020 and September 2025.

A total of 25 patients were included, most with B-cell lymphoma (76%, n=19) or multiple myeloma (24%, n=6). CMV reactivation occurred at a median of 16 days post CAR-T infusion, and maribavir was used as first-line therapy in 84% of patients, mainly due to cytopenias (80%), followed by toxicity (12%) or refractoriness (8%) to other antivirals.

Viral clearance was achieved in 100% of patients after a median of 8 days, with a median treatment duration of 14 days. No cases of CMV disease, CMV-related hospitalisation, or treatment failure were observed, and no maribavir-related toxicities were reported.

During follow-up, 12% of patients experienced CMV reactivation after initial clearance. Overall mortality was 28% (n=7), primarily due to progression of the underlying disease (16%), with additional deaths attributed to non-CMV infections (4%) and other causes (8%).

These findings suggest that maribavir may provide rapid and effective viral control with a favourable safety profile in CAR-T recipients, although results are limited by the small sample size.

TRIAL IN PROGRESS: A PHASE 2, SINGLE-ARM, MULTICENTER STUDY OF MARIBAVIR FOR CYTOMEGALOVIRUS INFECTION IN PATIENTS WITH LYMPHOID MALIGNANCIES UNDERGOING BISPECIFIC ANTIBODIES (MALMBA)

Soyean Kwon, *Boramae Medical Center Seoul, South Korea*
EBMT 2026, Poster P176

Maribavir is an oral, highly selective inhibitor of the CMV UL97 kinase. Unlike ganciclovir, cidofovir, and foscarnet—which are often limited by myelosuppression or nephrotoxicity—maribavir delivers strong antiviral activity without hematologic toxicity, making it suitable for immunocompromised patients. Bispecific antibodies (BsAbs) are an important treatment class for multiple myeloma (MM) and B cell lymphomas but increase infectious risk through immunoglobulin suppression, B cell depletion, cytopenias, and impaired cellular immunity. Clinically significant CMV reactivation or infection has been reported in up to 33% of MM patients treated with BsAbs, and CMV is a recognized risk factor for fatal infection in B cell lymphomas.

On this basis, an open label, single arm, multicenter study in Korea (NCT07014319) is evaluating the safety and feasibility of maribavir in patients with lymphoid malignancies who develop treatment emergent CMV infection or disease while receiving approved or investigational BsAbs.

The key study details are as follows:

- csCMVi is defined as CMV end organ disease or plasma CMV DNA ≥500 IU/mL on two consecutive tests and investigator assessment that treatment is necessary
- Eligible patients have adequate organ function and ability to undergo weekly virologic monitoring. Exclusions include severe renal or hepatic dysfunction, uncontrolled active viral infections, pregnancy, or any condition that prevent maribavir use
- Patients are treated with maribavir 400 mg orally twice daily from week 1 until CMV DNA is unquantifiable or falls below 500 IU/mL without CMV end organ disease.
- Quantitative plasma CMV DNA PCR is monitored on a weekly basis, additional diagnostics when clinically relevant

The endpoints are defined as:

- Primary endpoint is efficacy and safety of maribavir, including the proportion of patients achieving CMV DNA <500 IU/mL, time to reaching this threshold, and characterization of maribavir safety
- Secondary endpoints include time to >1 log10 reduction in CMV DNA, proportion of patients achieving that reduction, and rate of breakthrough CMV disease
- Exploratory endpoints include CMV specific cellular immunity to IE 1 and pp65 assessed at screening and end of treatment by T SPOT.CMV ELISpot (IFN γ-producing CD4+ and CD8+ T-cell spot counts per 250,000 PBMCs)

The MALMBA study is ongoing; two patients have been enrolled to date.

MULTI-VIRUS SPECIFIC MEMORY T CELLS TARGETING CMV, BKV, AND EBV IN POST-HSCT VIRAL INFECTIONS

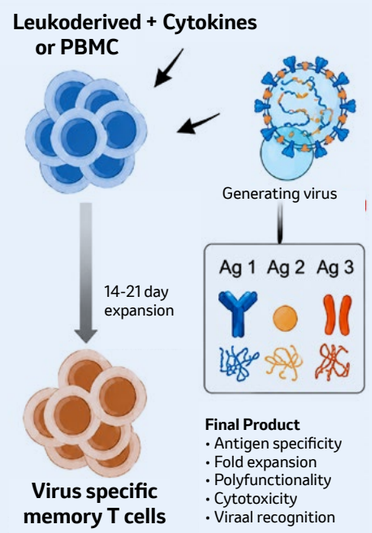
Seok-Goo Cho, *Research & Development division, LucasBio Co. Ltd. Seoul, South Korea*
EBMT 2026, Poster A270

Reactivation of CMV, BKV and EBV after transplantation can be life threatening. Antivirals exist for CMV but not for BKV or EBV, and current drugs are often limited by toxicity and drug resistance.

Virus specific T cell (VST) therapy represents a promising strategy. The experience with a SARS CoV 2 VST platform that resolved COVID 19 pneumonia in immunocompromised patients, provided the foundation for the development of multi-virus VST (Lee et al., Clin Infect Dis, 2025).
The authors developed an off-the-shelf, partially HLA-matched, multi-virus-specific memory T cell platform targeting CMV, BKV, and EBV by using peripheral blood mononuclear cells (PBMCs) from healthy donors. Virus-specific T cells were generated through peptide-based stimulation with immunodominant viral antigens, followed by ex vivo expansion. In vivo functional performance was evaluated in virus-relevant experimental models to support clinical translation. This platform is currently being evaluated in an ongoing Phase I/II clinical trial enrolling aHSCT recipients with antiviral-refractory or -resistant viral infections.

The final VST products showed strong, multi virus functional activity—including antigen specific IFN γ and TNF α release and cytotoxicity—without evidence of alloreactivity. Across multiple in vivo models, VST administration produced effective, target specific viral control with sustained T-cell expansion and persistence, increased infiltration of virus reactive CD8+ T cells at sites of infection.

Collectively, these findings demonstrated the functional potency and in vivo persistence of multi-virus-specific memory T cells, supporting their translational relevance for post-HSCT viral infections.



Adapted from Seok-Goo Cho, EBMT 2026, Poster A270

5. State of the art: diagnostics, monitoring & immune reconstitution

BEYOND THE PCR: CMV PNEUMONIA DESPITE NEGATIVE SERUM AND BRONCHIAL LAVAGE CMV VIRAL LOAD – CHALLENGE FOR ADVANCED NURSE PRACTITIONER

Hsin-Ju Yeh, *National Taiwan University Cancer Center* EBMT 2026, Poster NP69

CMV pneumonia can be a deadly complication in patients treated for hematologic disease, especially after aHSCT. Current pre-emptive strategies rely on monitoring CMV PCR viral load in serum or bronchoalveolar lavage (BAL). Though due to intracellular localization of CMV, tissue-invasive disease can be present even when extracellular viral loads are low or undetectable. This diagnostic mismatch is a challenge that can delay treatment and worsen outcomes. The authors reviewed 7 adult hematological patients (post-HSCT or chemotherapy) who developed CMV pneumonia despite low or negative CMV PCR in serum and/or BAL. Clinical severity, treatment timing and the use of combined therapy (ganciclovir and CMV hyperimmune globulin) were compared to extracellular viral loads. The results of the seven cases you find listed in the table below.

These cases illustrate that reliance on extracellular CMV viral load alone can be misleading. A negative serum or BAL PCR does not rule out tissue-invasive CMV pneumonia. Clinicians should integrate risk factors (like recent bendamustine use or lymphoid chemotherapy, steroid exposure, allogeneic HSCT and delayed immune recovery) and clinical/radiographic assessment into decision-making. Early multidisciplinary evaluation and prompt therapy may improve outcomes in these high-risk patients.

Case	Age/ Sex	Diag-nosis	Transplant / Status	CMV PCR (Serum)	CMV viral load (Bronchial wash)	Risk factor	Clinical Severity (Organ Involvement)	Key Treatment	Duration of Therapy	Outcome
1	58/M	DLBCL	10/10 Sibling PBSCT	Undetectable	Undetectable	Post CAR-T, immediate after Bendamustine	Severe Pneumonia	GCV + foscarnet + Cytotect (delayed)	4 weeks	Expired (Autopsy confirmed CMV inclusions)
2	74/F	AML	MUD PBSCT	Low (<1000 IU/ml)	Undetectable	Extensive chronic GVHD prolonged steroid use	Pneumonitis	Valcyte+ GCV + Cytotect + DLI Foscarnet	9 months	ARDS, Expired
3	70/F	T-LBL	Pre-HSCT (Post-Chemo)	Low (296 IU/ml)	Undetectable	Lymphoid malignancy, after steroid	Pneumonia, Severe GGO	GCV + Cytotect	~14 weeks	Recovered (GGO disappeared) connect to HSCT
4	56/M	AML	Post-Chemo (Relapsed)	High (15.5k IU/ml)	Undetectable	Steroid use for lung inflammation	Pneumonia	GCV + Cytotect	~6 weeks	Success / Recovered
5	31/F	Ph ALL	MUD PBSCT	Undetectable	Undetectable	Delayed immune reconstitution after HSCT	CNS/Retinitis/ Pneumonitis	GCV + Cytotect	N/A	Survived (Chronic complications)
6	64/F	AML	Haplo-PBSCT	Low (<1000 IU/ml)	Undetectable	Prolonged steroid use and immediate after pulse steroid for lung GVHD	Pneumonia	Valcyte+ GCV + Foscarnet Cytotect	Intermittent (~1 month)	Died of GVHD related infection
7	48/M	ATLL	MUD PBSCT	Undetectable	Undetectable VATS biopsy: CMV pneumonitis	Prolonged steroid use for grade III acute GVHD	Fatal Pneumonitis	Late GCV +Cytotect	N/A	Expired (CMV pneumonitis)

Adapted from Hsin-Ju Yeh. EBMT 2026, Poster NP69

RISK STRATIFICATION SYSTEM FOR CMV MONITORING AFTER AUTOLOGOUS HEMATOPOIETIC CELL TRANSPLANTATION

Alicja Sadowska-Klasa, *Fred Hutchinson Cancer Center, Seattle, United States* EBMT 2026, Poster B072

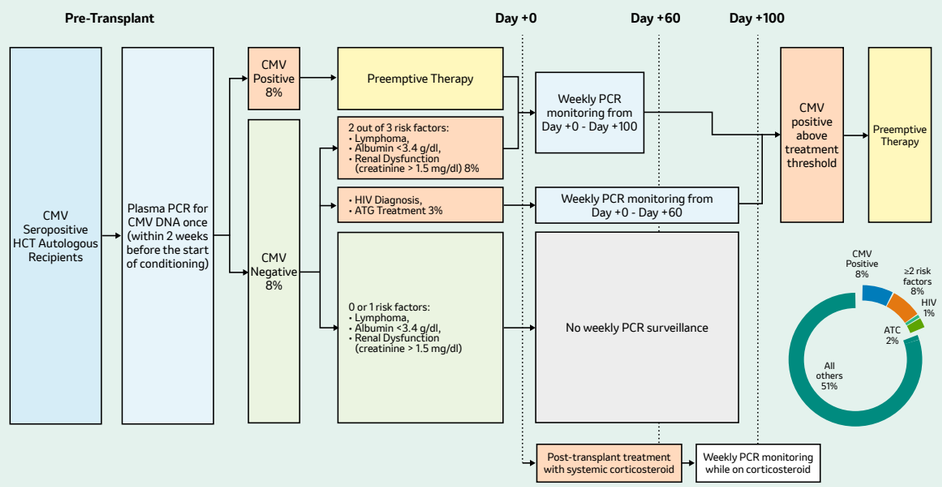
CMV monitoring is not routinely recommended after auto-HSCT, and its clinical relevance remains uncertain. This large retrospective single-centre study evaluated CMV reactivation patterns and developed a risk-based model to guide targeted monitoring in 1,000 CMV-seropositive auto-HSCT recipients transplanted between 2011 and 2021, with CMV monitoring performed between day +20 and +60.

By day 100, CMV viremia occurred in 29% of patients, while clinically significant CMV infection (csCMV) was observed in 12.7% and CMV disease in 1%. High-risk CMV infection occurred in 4.4% of patients and was more frequent in patients with lymphoma and in those receiving ATG. CMV serostatus did not affect NRM at day 100 or OS at 3 years.

In univariable analyses, pre-transplant CMV viremia was associated with an increased risk of post-transplant CMV infection (HR 11.6; 95% CI 3.63–36.8), while HIV infection was associated with CMV disease (HR 16.1; 95% CI 2.03–128). In multivariable analysis, elevated pre-transplant creatinine (>1.5 mg/dL), hypoalbuminemia (<3.4 g/dL), and lymphoma diagnosis remained significant predictors of high-risk CMV reactivation, while post-transplant steroid use (>0.5 mg/kg) further increased this risk (HR 4.7; 95% CI 1.86–11.9).

A risk model incorporating these factors stratified patients by incidence of high-risk infection, ranging from 1.4% in patients without risk factors to 9.4% in those with ≥2 risk factors, increasing to 30% when pre-transplant CMV viremia was included

These findings support a risk-adapted CMV monitoring strategy after auto-HSCT, focusing surveillance on patients with predefined clinical risk factors rather than universal screening.



Adapted from A. Sadowska-Klasa EBMT 2026, Poster B072

EARLY ADMINISTRATION OF LETERMOVIR PROPHYLAXIS POST-ALLOGENEIC HEMATOPOIETIC CELL TRANSPLANTATION IS ASSOCIATED WITH DELAYED ENGRAFTMENT

Léna Royston, *Geneva University Hospitals Geneva, Switzerland*
EBMT 2026, Poster B099

Letermovir prophylaxis has substantially reduced CMV-related complications after aHSCT, but its impact on hematopoietic recovery remains unclear. This single-centre retrospective cohort study evaluated engraftment and clinical outcomes in the pre- and post-letermovir era, including patients transplanted between November 2015 and December 2023. Letermovir prophylaxis was introduced in May 2019 and administered from day 1 to day 100 post-transplant in CMV-seropositive recipients.

A total of 137 and 177 CMV-seropositive patients were included in the pre- and post-letermovir periods, respectively. Baseline characteristics were largely comparable, although the letermovir period included more peripheral blood stem cell grafts and post-transplant cyclophosphamide-based GVHD prophylaxis.

Engraftment was slightly but significantly delayed in the letermovir period (median 18 vs 17 days; p=0.009). In multivariable analysis, letermovir prophylaxis was independently associated with delayed engraftment (HR 0.71; p=0.008).

At the same time, 1-year all-cause mortality was significantly lower in the letermovir period (p<0.001), while 1-year incidence of acute GVHD and hematologic relapse was comparable between groups.

These findings suggest that, despite improved survival outcomes, letermovir prophylaxis may be associated with a slight but significant delay in engraftment, the clinical relevance of which remains to be determined

IMPACT OF DONOR CMV-SPECIFIC HUMORAL AND CELL-MEDIATED IMMUNITY ON CMV-IMMUNE RECONSTITUTION AND INFECTION AFTER ALLOGENEIC HEMATOPOIETIC STEM-CELL TRANSPLANTATION

Guillermo Ramos Moreno, *Hospital 12 de Octubre, Madrid, Spain*
EBMT 2026, Poster B108

CMV risk after allogeneic HSCT is largely driven by donor/recipient serostatus. This prospective cohort study of 127 consecutive aHSCT recipients examined how donor CMV serology and CMV-specific cell-mediated immunity (measured by QuantiFERON®-CMV IFN-γ release assay, QTF-CMV) affected immune reconstitution and csCMVi. Donor and recipient CMV-IgG serology was measured pre-transplant; QTF-CMV was measured monthly till 6 months after transplant. csCMVi was defined as CMV DNAemia requiring pre-emptive therapy and/or CMV disease.

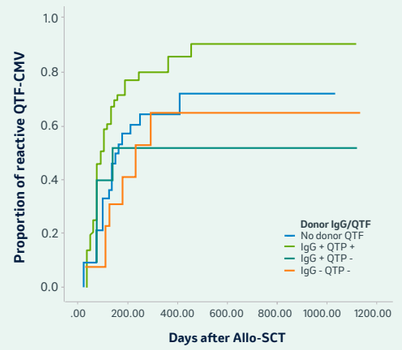
Most patients received PTCy-based GVHD prophylaxis (94%) and 53% received primary CMV-prophylaxis with letermovir. The overall incidence of CMV reactivation was 63%. Letermovir prophylaxis significantly reduced the incidence of csCMVi (18% vs 25%, p< 0,05), although it did not have a significant impact on NRM or OS in this population.

The key findings regarding CMV-CMI were:

- Cumulative incidence of QTF-CMV rose over time: 19.6% at month +1 (20/102), 42.8% at month +3 (30/70), 52.5% at month +6 (21/40)
- Recipients of CMV IgG+ donors had higher median IFN-γ at month +3 (0.17 vs 0.00 IU/mL; p=0.025) and +6 (0.54 vs 0.01 IU/mL; p=0.003) compared to IgG- donors
- Pre-transplant QTF-CMV was done in 84 related donors: among the CMV IgG+ donors, 84.3% (59/70) had a reactive QTF-CMV assay (QTF-CMVR)
- Letermovir use was similar between both groups (55,7% in IgG+ QTF-CMVR, 54% in IgG+ QTF-CMVNR and 64% in IgG- donors)
- All recipients of IgG+ QTF-nonreactive donors were CMV IgG+; 91.4% of recipients of IgG+ QTF-reactive donors were IgG+ (p=0.5)
- Recipients from QTF-reactive donors showed higher IFN-γ at month +1 (0.01 vs 0.00 IU/mL; p=0.057), +3 (0.15 vs 0.00; p=0.086) and +6 (1.48 vs 0.02; p=0.038) versus QTF-nonreactive donors
- csCMVi incidence tended to be lower with IgG+ QTF-reactive donors at +3 and +6 (37.7% vs 63.3%; p=0.107 and 41.5% vs 72.7%; P=0.059 respectively)
- A higher proportion of recipients showed QTF reactivity during follow-up when their donor was IgG+/QTF-reactive versus IgG+/QTF-nonreactive (78% vs 45%).
- No differences were seen in overall mortality or GVHD between QTF-reactive and nonreactive donor groups.

The authors conclude donor CMV-specific humoral and cellular immunity contributed to earlier CMV-CMI reconstitution after aHSCT and thus reduced csCMVi risk. These findings may inform donor selection and graft considerations in the future.

Evolution of CMV QTF reactivity by donor CMV inmune status



Adapted from G. R. Moreno EBMT 2026, Poster B108

NOT ALL AUTOLOGOUS TRANSPLANTS ARE LOW RISK: DELAYED PLATELET ENGRAFTMENT SIGNALS THE NEED FOR CMV MONITORING

Ibrahim Ethem Pinar, *Bursa Uludag University, Bursa, Turkey*
EBMT 2026, Poster B123

CMV monitoring is not routinely recommended after auto-HSCT, although the risk of reactivation appears heterogeneous. This multicentre retrospective study evaluated risk factors for CMV reactivation to support a risk-adapted monitoring strategy.

A total of 614 adult patients undergoing auto-HSCT were analyzed, of whom 12.5% (n=77) developed CMV reactivation. Baseline characteristics and laboratory parameters were comparable between patients with and without reactivation. Hematopoietic recovery kinetics differed between groups. Patients with CMV reactivation had significant delayed platelet engraftment (median 14 vs 12 days; p<0.001) and modestly delayed neutrophil engraftment (12 vs 11 days; p=0.017). Advanced disease stage (p=0.012), prior rituximab exposure (p=0.008), and comorbidities (p=0.027) were more frequent in patients with CMV reactivation.

In multivariable analysis, delayed platelet engraftment (≥16 days) was the only independent predictor of CMV reactivation (OR 2.4; 95% CI 1.5–3.9; p<0.001).

These findings confirm that CMV risk after auto-HSCT is not uniform and support a targeted monitoring approach, in which patients with delayed platelet recovery may benefit from CMV PCR surveillance.

ACCURATE CMV IGG DETECTION USING BUCCAL SWABS IN STEM CELL DONOR SCREENING

Paula Juan-Pardo, Poster Presenter
Alejandra Martinez Trillos, Centro Diagnostico Biomedico, Hospital Clinic Barcelona, Spain
EBMT 2026, Poster B140

The assessment of CMV serostatus is essential for donor selection, as a mismatch increases the risk of viral reactivation and survival . Standard CMV IgG testing requires venipuncture, which can hinder rapid triage, early donor registration and selection, or mass screening. Buccal swab–based serology could be a minimally invasive alternative, but comparative data versus validated serum assays are limited.

This study compared buccal swab CMV IgG (DRG ELISA) to serum CMV IgG (Elecsys) in 222 paired donor samples. Discordant pairs were retested; reproducibility was checked by repeating swabs at 1 and 3 months after initial collection.

Among paired samples, 115 had concordant positive results in both serum and buccal swab (51.8%), and 91 were negative (41.0%). Five samples (2.3%) were positive in serum but negative in swab, considered potential false negatives. Eleven samples (5.0%) were positive in swab but negative in serum; seven corresponded to grey zone values and four were clearly positive. Two discrepancies were resolved upon retesting, five maintained their initial results.
The diagnostic performance metrics for CMV IgG detection using buccal swabs showed sensitivity of 96.8%, specificity 90.1%, PPV 92.1%, NPV 95.8%, and overall accuracy 93.7%. Cohen's Kappa was 0.873, indicating almost perfect agreement between the two methodologies.
Reproducibility analysis showed consistent results in 50/54 buccal swabs (92.6%) at one month and in 28/28 samples (100%) at three months after sample's collection.

In conclusion, buccal swab CMV IgG testing showed excellent concordance, high accuracy, and strong reproducibility versus serum. It is a reliable, less invasive option for rapid or large-scale donor CMV screening.

LETERMOVIR PROPHYLAXIS RESHAPES EARLY IMMUNE RECONSTITUTION AND CMV/EBV REACTIVATION DYNAMICS AFTER ALLOGENEIC HSCT

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EBMT 2026, Poster B190

Since letermovir prophylaxis redefined CMV management after aHSCT, its effects on early immune recovery remain underexplored. The researchers retrospectively analyzed immune reconstitution patterns in 148 patients (prophylaxis n=81, pre-emptive n=67, treated between April 2021 en December 2024). Both clinical and virologic data was assessed, peripheral blood lymphocyte subsets by flow cytometry were measured at baseline and monthly for 12 months.

At a median follow-up of 28.1 months (range, 6.5–49.1), 2-year OS (83.6% vs 82.4%) and NRM (10.5% vs 10.1%) were similar between both groups (p>0.05). Within 180 days, prophylaxis greatly reduced csCMVi (61.2% vs 12.4%, p<0.001), delayed CMV activation (median 25 vs 122 days, p<0.001), and shortened infection duration (34 vs 19 days, p=0.017). Peak viral load and CMV disease rates remained similar between groups (p=0.240 and p=0.341 respectively). EBV activation trended higher with letermovir (14.9% vs 27.2%, p=0.083) but duration, peak load, and PTLD incidence were comparable.
When using a competing-risk analysis, letermovir independently reduced csCMVi (HR 0.16, 95% CI 0.08–0.35, p<0001), while clinically significant csEBV infection was independently associated with rATG exposure (HR 2.50) and prophylaxis (HR 2.63).
Though baseline immune profiles were comparable, the prophylaxis group had significantly lower total lymphocytes, CD3+, CD8+ T cells, NKT cells in counts and percentages, CD4+ T-cell absolute counts, and NK-cell percentages (all p< 0.05), (+2 to +6 months post-transplant). The proportion of B-cells were consistently higher; these differences resolved after month +6.

In a later stage, letermovir recipients exhibited a distinct T-cell maturation profile; including higher CD4+ T-cell Treg proportions and CD4/CD8 ratios (p<0.05). The percentages of CD4+ and CD8+ naïve T cells, central memory (TCM) subsets, and CD8+ TEMRA subsets were significantly increased, whereas effector memory (TEM) proportions in both CD4+ and CD8+ compartments were significantly reduced (p< 0.05).

In summary, letermovir prophylaxis recipients showed an unique immune reconstitution profile, highlighted by early T-cell suppression. By avoiding CMV-driven immune activation and exhaustion, T-cell TCR diversity was retained, preserving robust T-cell reserve capacity.

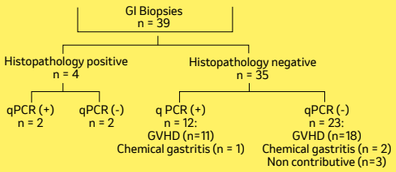
DIAGNOSTIC VALUE OF CMV PCR ON GASTROINTESTINAL BIOPSIES IN ALLOGENEIC HEMATOPOIETIC STEM CELL TRANSPLANT RECIPIENTS

Sabrine Khirallah, *National Bone Marrow Transplantation Center, Tunis, Tunisia*
EBMT 2026, Poster P159

Diagnosis of CMV gastrointestinal (GI) disease after allogeneic HSCT is commonly based on histopathology and plasma CMV PCR, but the sensitivity might be limited. This descriptive study retrospectively assessed whether quantitative CMV PCR on GI biopsy tissue adds diagnostic value.

aHSCT recipients from HLA-matched sibling or haploidentical donors (2021 - 2025) were included who underwent GI biopsy for suspected CMV GI disease. CMV was monitored in plasma by RT-qPCR from engraftment until day +100. Patients with suspected CMV GI disease underwent simultaneous GI biopsy sampling for both histopathological assessment and quantitative CMV PCR testing performed on the same specimen. Histological diagnosis of CMV GI disease was based on the existence of intranuclear and/or cytoplasmic viral inclusions.

In total 39 biopsies from 25 patients were obtained; 84% was CMV-seropositive at baseline. GVHD occurred in 92% of patients and preceded CMV reactivation/suspected GI disease in 88% (22/25). The results of histopathology are reflected in this figure.



In this analysis, CMV PCR on GI biopsy specimens provided complementary diagnostic value to histopathology and plasma PCR. CMV PCR detected viral DNA in biopsies that were histologically negative, including cases with negative or low-level plasma viremia. Using tissue CMV PCR might improve detection and timely antiviral therapy after aHSCT.

6. Case reports

FOSCARNET SODIUM ENEMA FOR REFRACTORY CMV GASTROENTERITIS AFTER HAPLOIDENTICAL HSCT: A CASE SERIES

Mengjing Li, *The first affiliated hospital, Zhejiang University School of Medical Hangzhou, China*
EBMT 2026, Poster NP26

CMV gastroenteritis is a serious complication after allogeneic HSCT that can persist despite systemic antiviral therapy. In two cases of haploidentical HSCT recipients it was assessed whether rectal foscarnet sodium enemas, with or without intravenous foscarnet, could reduce intestinal CMV viral load and speed symptom resolution in these situations. Both patients with refractory CMV gastroenteritis and positive stool CMV-DNA received foscarnet retention enemas as salvage or adjunctive therapy. Stool/blood viral loads and symptoms were monitored. Stool CMV-DNA was cleared rapidly (within days) and produced marked symptom improvement.

In these case studies foscarnet sodium enema appeared to be a well-tolerated, non-invasive adjunct that reduced local viral burden and accelerated symptom resolution in refractory CMV gastroenteritis after aHSCT. Evaluation in larger studies is needed.

HEMATOPOIETIC STEM CELL TRANSPLANTATION WITHOUT CONDITIONING IN IL2RG-MUTATED SCID AND SEVERE ACTIVE CMV INFECTION: RAPID IMMUNE RECONSTITUTION AND INFECTION CONTROL

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EBMT 2026, Poster P269

Active CMV infection prior to HSCT is associated with poor outcomes in patients with SCID. This case report, based on a retrospective chart review, describes the outcome of an unconditioned HSCT in an infant with IL2RG-mutated SCID and severe CMV infection.

A 4-month-old patient presented with disseminated CMV infection, including severe encephalitis and retinitis, with persistent high viral load despite intensive antiviral therapy with foscarnet and ganciclovir. HSCT was performed at 6 months using an unmanipulated bone marrow graft from a matched sibling donor, without conditioning, while CMV replication was ongoing.

Rapid immune reconstitution was observed, with naïve CD4+ T cells detected by day +8 and CMV PCR becoming negative by day +24. Donor chimerism was dominant by day +21 (62%), and the patient was discharged at day +31. The patient remained free of GVHD and severe infections, with sustained donor chimerism and immune recovery during 24 months of follow-up.

This case illustrates that unconditioned HSCT can enable rapid immune reconstitution and viral control in SCID patients with active CMV infection, particularly when a matched sibling donor is available. It may allow infection control while avoiding conditioning-related toxicity, in line with current guideline recommendations.

MARIBAVIR TREATMENT IN AGGRESSIVE EBV-ASSOCIATED POSTTRANSPLANT LYMPHOPROLIFERATIVE DISORDER (EBV+ PTLD)

Wei-Han Huang, *Hualien Tzu Chi Hospital Hualien, Taiwan*
EBMT 2026, Poster P287

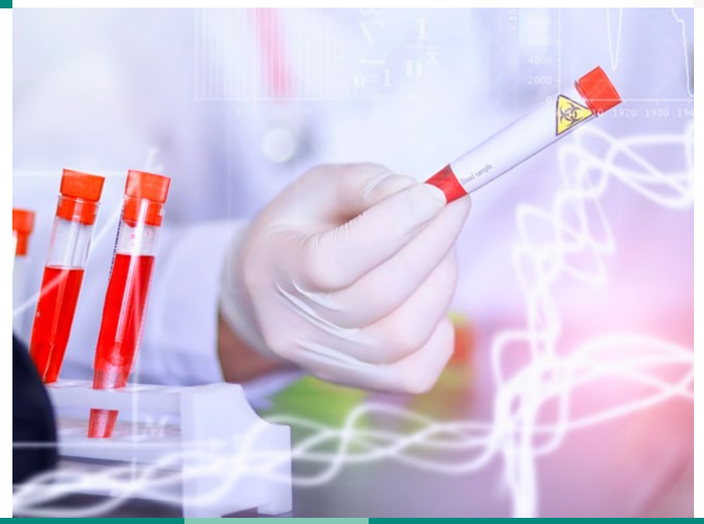
EBV+ PTLD after aHSCT has variable outcomes depending on factors as histology, stage, and ability to reduce immunosuppression; standard treatment often uses immunosuppression reduction (RIS) and R CHOP chemoimmunotherapy.
Maribavir inhibits the EBV BGLF4 viral kinase and has been shown experimentally to reduce viral genome copies and infectivity.

In this case report, the authors describe a unique patient case of a 53 year old woman, with HTLV 1-associated ATLL (stage IV) treated in 2023 with multiagent chemotherapy and intrathecal therapy, followed by reduced intensity allogeneic PBSCT from an 8/8 matched unrelated donor. Early post transplant complications included PJP and a rapid taper of GVHD prophylaxis.

In June 2025 her disease relapsed with extramedullary nodal lesions; and she received multiple salvage therapies and two DLI infusions. She then developed severe liver and GI GVHD requiring broad immunosuppression (steroids, tacrolimus, mycophenolate, abatacept, ATG and ruxolitinib). Post discharge she was readmitted with an E. coli urosepsis, carbapenem resistant A. baumannii pneumonia, a Candida dubliniensis infection, requiring ICU care with hemodialysis, and mechanical ventilation.

In September 2025, progressive neck lymphadenopathy developed with intermittent fever and rising EBV viral load. Excisional biopsy confirmed EBV+ PTLD (EBER positive). Management comprised of reducing immunosuppression, maribavir daily for three weeks, single-dose Ropeginterferon, and one DLI infusion. The EBV load fell from 23,300 to 2,000 IU/mL with concurrent lymphoma regression. The patient continued on low dose tacrolimus and ruxolitinib for ongoing GI GVHD and is currently disease free.

Significance: about half of EBV+ PTLDs relapse or resist initial RIS or rituximab. While maribavirs efficacy is not proven in clinical trials for EBV+ PTLD, this case demonstrated virologic and clinical response in a highly immunosuppressed, complicated post transplant patient. The observed effect aligns with maribavir's inhibition of BGLF4, which is required for the EBV lytic replication and implicated in lymphomagenesis. This supports further investigation of BGLF4 inhibition as a therapeutic strategy.



Abbreviations

ACV	acyclovir
AdV	adenovirus
AE	adverse event
ALC	absolute lymphocyte count
ALL	acute lymphoblastic leukemia
AML	acute myeloid leukemia
(a-)HSCT	(allogeneic) hematopoietic stem cell transplantation
auto-HSCT	autologousc hematopoietic stem cell transplantation
ASTCT	American Society for Transplantation and Cellular Therapy
ATG	anti-thymocyte globulin
BAL	bronchoalveolar lavage
BiTE	bispecific T-cell engager
BIW	twice weekly (bis in week) — dosing schedule
BGLF4	beta/gamma-like protein kinase 4
BK	BK polyomavirus
BM	bone marrow
BsAbs	bispecific antibodies
CAR-T	chimeric antigen receptor T-cell
CB(T)	cord blood (transplantation)
CI	confidence interval
CIBMTR	Center for International Blood and Marrow Transplant Research (registry)
CIR	cumulative incidence of relapse
CKD	chronic kidney disease
CMI	cellular mediated immune (response)
CMV	cytomegalovirus
CMVd	cytomegalovirus disease
CMV-CMI	CMV cell-mediated immunity
CMV-IgG	CMV immunoglobulin
CNS	central nervous system
CRS	cytokine release syndrome
CTIWP	cellular therapy & immunobiology working party (of the EBMT)
(cs-)CMVi	(clinically significant) CMV infections
CTL	cytotoxic T-cell
DAH/IPS	diffuse alveolar hemorrhage/idiopathic ineumonia syndrome
DFS	disease free survival
DLI	donor lymphocyte infusion
DOM	disorders of metabolism
EBMT	European Society for Blood and Marrow Transplantation
(cs)EBV	(clinically significant) Epstein-Barr virus
EBV-R	Epstein-Barr virus reactivation
ECIL	European Conference on Infections in Leukemia
ELISpot	enzyme-linked immunospot (assay)
(a)(c)GVHD	(acute) (chronic) graft versus host disease
FOS	foscarnet
GETH-TC	Grupo Español de Trasplante Hematopoyétic
GI	gastrointestinal (tract)
G-CSF	granulocyte-macrophage colony-stimulating factor
GCV	ganciclovir
GVHD	graft versus host disease, (a) acute or (c) chronic
haplo-HSCT	HLA-haploid hematopoietic stem cell transplantation
HBMT	haploidentical blood and marrow transplant
HCRU	healthcare resource utilization
HCV	hepatitis B virus
HLA	human leukocyte antigen
HSCT	hematopoietic cell transplantation
HHV	human herpes virus
HSV	herpes simplex virus
HR	hazard ratio
IAT	investigator assigned therapy
IC50 / EC50	effective concentration (50%) / half-maximal effective concentration (pharmacology; EC50 cited for phenotypic resistance)
ICANS	immune effector cell-associated neurotoxicity syndrome
IDWP	Infectious Diseases Working Party (of the EBMT)
IEWP	Inborn Errors Working Party (of the EBMT)
IFI	invasive fungal infection
IFN(-y)	interferon gamma
IgG	immunoglobulin
IU	international units
IVIG	intravenous immunoglobulin
LBL	lymphoblastic blastoma
LLOQ	lower limit of quantification (laboratory assay metric)
LWP	lymphoma working party
MDS	myelodysplastic syndromes
MM	multiple myeloma
MMF	mycophenolate mofetil
MRD	matched related donor
MUD	matched unrelated donor
MMUD	mismatched unrelated donor
MSD	matched sibling donor
NK(T)	natural killer T-cell
NRM	non-relapse mortality
OAT1	organic anion transporter 1
OS	overall survival
OTUS	outcomes, treatment patterns, and utilization study
PB	peripheral blood
PBSC	peripheral blood stem cell
PBMC	peripheral blood mononuclear cells
PCR	CMV (quantitative) polymerase chain reaction
PDWP	Pediatric Diseases Working Party (of the EBMT)
PET	pre-emptive therapy
PFS	progression free survival
PID	primary Immunodeficiency
PTLD	post-transplant lymphoproliferative disorder
PFS	progression free survival
PTCy	post-transplant cyclophosphamide
QNAT	quantitative nucleic acid testing
(q)PCR	(quantitative) polymerase chain reaction
QTF-CMV	QuantiFERON-CMV
(r)ATG	(rabbit) anti-thymocyte globulin
RBC	red blood cell
RCT	randomized controlled trial
RIC	reduced intensity conditioning
RIS	radiologically isolated syndrome
RR	refractory or resistant
RRI	refractory, resistant or intolerant
RNA	ribonucleïnezuur
RSV	respiratory syncytial virus
SCID	severe combined immunodeficiency
SOS	sinusoidal obstruction syndrome
SOT	solid organ transplant
S(m)PC	summary of product characteristics
TAE	treatment emergent adverse events
TA-TMA	transplant-associated thrombotic microangiopathy
TBI	total body irradiation
TDM	routine drug monitoring
T-cell	thymus derived lymphocyte
TCR	T-cell receptor
Treg	regulatory T-cell
TCM	central memory T-cells
TEM	effector memory T-cells
TEMRA	terminally differentiated effector memory T-cells re-expressing CD45RA
UD	unrelated donor
VGCV	valganciclovir
VL	viral load
VOD	veno-occlusive disease
VST	virus-specific T lymphocytes
VZV	varicella-zoster virus (=HHV-3)

Colophon

Editorial:
Hanka Verburg, MSD
Jeanette Scholder, MSD
Hester van der Velden, DM Farma Communicatie

Production:
Frans Dielissen and team, DM Farma Communicatie

Special thanks to:
Preston Ray Pearson, EBMT Marketing Manager

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NL-NON-03491 May 2026