



Article

Maribavir for Refractory Cytomegalovirus Infection in Transplant Recipients: A Single-Center Real-World Case Series with Tailored Therapeutic Management

Biagio Pinchera ^{1,*} , Alessia D'Agostino ¹, Stefano Mario Susini ¹, Maria Michela Scirocco ¹, Fabrizio Salemi ², Amerigo Piccione ², Rosa Carrano ² and Ivan Gentile ¹

¹ Division of Infectious Diseases, Department of Clinical Medicine and Surgery, University of Naples "Federico II", 80131 Naples, Italy; alessiadagostino53@gmail.com (A.D.); stefano.susini1@gmail.com (S.M.S.); mmichelascirocco@hotmail.com (M.M.S.)

² Division of Nephrology, Department of Public Health, University of Naples "Federico II", 80131 Naples, Italy; fabriziosalemi@yahoo.it (F.S.); amerigopiccione@libero.it (A.P.); rosa.carrano@alice.it (R.C.)

* Correspondence: biapin89@virgilio.it

Abstract

Background: Refractory cytomegalovirus (CMV) infection remains a major challenge after solid organ transplantation (SOT) and hematopoietic stem cell transplantation (HSCT). Maribavir has demonstrated efficacy and a favorable safety profile in clinical trials, but real-world data on virological endpoints, treatment duration, and recurrence remain limited. **Methods:** We retrospectively reviewed all consecutive transplant recipients treated with maribavir for refractory CMV infection/disease at a tertiary referral center in Naples, Italy. Clinical and laboratory information were extracted from medical records. **Results:** Six patients were included: five SOT recipients and one autologous HSCT recipient. Maribavir was administered at 400 mg twice daily for a median of 43.5 days (IQR 23–56 days). At the end of treatment, five patients had detectable CMV DNA < 1000 IU/mL without confirmed clearance. Patient 6 was the only patient to achieve confirmed clearance (<LLOQ on two consecutive tests) and experienced virological and clinical rebound 18 days after discontinuation. Overall, 3/6 patients rebounded within 15–21 days. Adverse events were mild, and no patient discontinued therapy because of toxicity. **Conclusions:** In this small real-world series, maribavir was well tolerated and was associated with marked reductions in CMV DNA. However, three patients experienced early post-treatment rebound, including the only patient who achieved confirmed clearance. These observations are hypothesis-generating and do not establish a stopping threshold for low-level DNAemia or a validated post-treatment monitoring interval. Larger studies incorporating longitudinal viral kinetics, immune recovery, and resistance testing are needed.



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1. Introduction

CMV is a leading cause of infectious morbidity after SOT and HSCT and continues to shape post-transplant outcomes through both direct and indirect effects. Direct effects include CMV syndrome and tissue-invasive disease, while indirect effects include acute and chronic allograft dysfunction, increased risk of secondary infections, and immune

dysregulation. In SOT, risk is strongly influenced by donor–recipient serostatus mismatch and intensity of immunosuppression; in HSCT, it is driven by conditioning regimen, graft source, graft-versus-host disease, and immune reconstitution dynamics. Despite decades of advances in virology, immunology, and antiviral therapy, CMV continues to represent an “evergreen” challenge in transplantation [1–4].

Prevention relies on prophylaxis and/or pre-emptive therapy guided by serial CMV DNA monitoring. However, when established CMV infection/disease requires treatment, available therapeutics historically center on DNA polymerase inhibitors—ganciclovir/valganciclovir, foscarnet, and cidofovir. These agents are effective but limited by toxicity: myelotoxicity with (val)ganciclovir, and nephrotoxicity/electrolyte disturbances with foscarnet and cidofovir. In practice, toxicity often forces dose reductions or early discontinuation, resulting in suboptimal antiviral exposure that may favor persistent replication and resistance selection.

R/R CMV has therefore emerged as a major clinical challenge. Updated consensus definitions distinguish refractory CMV (persistent or rising CMV DNAemia despite adequate antiviral exposure) from resistant CMV (refractoriness plus documented genotypic mutations that reduce susceptibility), and propose standardized criteria for describing viral kinetics, exposure, and outcomes [1]. These definitions are especially relevant to real-world series, where treatment decisions depend on longitudinal trajectories rather than on single measurements.

The clinical and economic impact of R/R CMV is substantial. Patients often require prolonged hospitalization, intravenous antivirals, intensive laboratory monitoring, and repeated imaging or endoscopic evaluations. In SOT, uncontrolled CMV can precipitate acute allograft dysfunction and may contribute to chronic injury; in HSCT, CMV complicates graft-versus-host disease management and increases mortality risk. Because R/R CMV typically develops in patients with the most intense immunosuppression, it frequently co-exists with other opportunistic infections and complicates the overall post-transplant course. These considerations make it critical not only to achieve short-term viral reduction, but to sustain control while minimizing drug toxicity and preserving future antiviral options [2–4].

A second, often underappreciated, challenge is assay interpretation at low viral loads. Modern quantitative PCR assays are highly sensitive, but variability near the LLOQ can create uncertainty around whether a low-level signal represents clinically meaningful replication or assay noise. This uncertainty is precisely what complicates stopping decisions under maribavir therapy: clinicians may be reluctant to continue therapy indefinitely for “trace” DNAemia, yet premature discontinuation may allow rapid regrowth if replication persists. Therefore, an explicit strategy for low-level DNAemia—defining what constitutes a plateau, when to test for resistance, and how to monitor after stopping—is central to real-world maribavir stewardship [1,5,6].

Maribavir is an oral UL97 kinase inhibitor with a mechanism of action distinct from polymerase inhibitors. Regulatory approval for post-transplant R/R CMV infection/disease was primarily based on the phase 3 SOLSTICE trial, which demonstrated superior confirmed viremia clearance at week 8 compared with investigator-assigned therapy, with a safety profile dominated by dysgeusia rather than nephrotoxicity or myelosuppression [5,6]. The oral route and toxicity-sparing profile make maribavir especially attractive for prolonged outpatient management [7,8].

Yet several practical questions remain insufficiently answered by trial endpoints. Clinicians may observe low-level CMV DNAemia during maribavir therapy and must decide whether to continue, stop, or switch treatment. Relapse after discontinuation may reflect several non-mutually exclusive factors, including residual replication, incomplete immune

recovery, ongoing immunosuppression, variability in antiviral exposure or adherence, assay variability near the LLOQ, or antiviral resistance. These mechanisms cannot be distinguished without patient-level pharmacologic, immunologic, and genotypic data [6,9–12]. Product information also reports lower response rates in patients with high baseline CMV DNA ($\geq 50,000$ IU/mL), suggesting that baseline viral load may be relevant when interpreting outcomes, but it does not define an individual stopping rule [6].

In this context, real-world case series remain valuable because they describe decision points that may not be captured by randomized trial endpoints. Here, we present six patients (five SOT recipients and one autologous HSCT recipient) treated with maribavir for refractory CMV, with attention to safety, viral kinetics, and early post-treatment rebound. Given the small sample size and retrospective design, these observations are intended to generate hypotheses rather than define stopping rules, monitoring schedules, or treatment algorithms.

2. Materials and Methods

We conducted a retrospective observational case series at the Infectious Diseases Unit of the University Hospital “Federico II” (Naples, Italy). All consecutive transplant recipients (SOT or HSCT) treated with maribavir for refractory CMV infection/disease were eligible. Eligibility and terminology were aligned with the 2024 Transplant-Associated Virus Infections Forum consensus update [1]. Genotypic resistance was not required for inclusion; available UL97/UL54 results, if any, were recorded on a per-patient basis.

Cases were identified through pharmacy dispensing records and infectious diseases consultation logs. Maribavir was prescribed at 400 mg twice daily, consistent with product information [6,7]. Treatment decisions—including initiation timing, adjunctive measures, and immunosuppression adjustments—were individualized by the transplant and infectious diseases teams, based on clinical severity, baseline viral load, prior antiviral exposure/toxicity, and kidney function.

Adjunctive measures were not standardized. Some patients received short “bridging” with foscarnet during the first three maribavir days and/or anti-CMV immunoglobulin as an early intensification strategy. The decision to use foscarnet was guided by creatinine clearance (CrCl). Foscarnet was considered when CrCl was ≥ 0.4 mL/min/kg, whereas CMV-specific immunoglobulins were preferred when CrCl was < 0.4 mL/min/kg. Immunosuppression reduction (for example, temporary reduction in antimetabolites or calcineurin inhibitor exposure) was pursued when clinically feasible, consistent with common guideline approaches [3,4].

In our center, CMV DNA was generally monitored at least weekly during active therapy, with more frequent testing in selected high-risk situations. After treatment discontinuation, monitoring was individualized. This retrospective series was not designed to compare monitoring schedules or validate a specific monitoring interval.

Because drug–drug interactions are clinically relevant for maribavir, therapeutic drug monitoring for calcineurin inhibitors and mTOR inhibitors was routinely performed after maribavir initiation, and subsequent doses were adjusted when clinically indicated [6]. Adverse events were assessed by chart review, including patient-reported dysgeusia, gastrointestinal symptoms, laboratory abnormalities (serum creatinine and electrolytes), and hematologic parameters.

We abstracted demographics, transplant type, immunosuppressive regimen, prior anti-CMV therapies (agent, dose, duration, and reason for change), adjunctive interventions, maribavir duration, adverse events, longitudinal CMV DNA results, post-maribavir follow-up, management of rebound, resistance testing at rebound, and final clinical and virological outcome from electronic medical records. Because this was routine care, geno-

typic resistance testing (UL97/UL54) was not performed systematically; when available, results were recorded.

Plasma CMV DNA was monitored using the Roche platform in routine clinical care and reported as international units per milliliter (IU/mL). We used the following operational definitions to describe response patterns:

- Viremia clearance (local operational definition): CMV DNA below the lower limit of quantification (LLOQ) (<100 IU/mL) on two consecutive tests collected ≥ 24 h apart.
- Low-level DNAemia: detectable CMV DNA < 1000 IU/mL.
- Low-level DNAemia plateau: persistent low-level DNAemia with <0.5 log₁₀ decline over ≥ 14 days despite ongoing maribavir.
- Early post-treatment rebound: renewed CMV DNA increase requiring additional anti-CMV therapy within 4 weeks after maribavir discontinuation.
- Complete clinical response (local operational definition): resolution of all signs and symptoms attributed to CMV and, when applicable, no ongoing clinical evidence of tissue-invasive CMV disease at the end of treatment. Patients treated for asymptomatic DNAemia were categorized as clinically stable rather than as having a complete clinical response.

Consensus documents use explicit antiviral-exposure and log₁₀ trajectory criteria for refractoriness and may use stricter clearance definitions [1,5,6]. We therefore report the operational definitions above and patient-level data wherever available. Given the sample size, all analyses are descriptive and no stopping threshold, monitoring interval, or causal mechanism was prespecified or tested.

The primary descriptive outcomes were: (i) virological response (decline to <1000 IU/mL and/or confirmed clearance), (ii) early rebound after discontinuation, and (iii) maribavir tolerability.

This study used retrospective, routinely collected clinical data. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and good clinical practice and was approved by the Ethics Committee of A.O.U. “Federico II” of Naples (protocol 37/2025). The authors confirm that the ethical policies of the journal were observed.

Given the small sample size, analyses were purely descriptive, and no formal statistical comparisons were performed.

3. Results

Six transplant recipients received maribavir for refractory CMV infection/disease (Table 1): five were kidney SOT recipients and one was an autologous HSCT recipient. Five patients were female; the median age was 49.5 years (IQR 33–58). All patients had persistent or rising CMV DNAemia after prior anti-CMV therapy.

Baseline CMV DNA at maribavir initiation ranged from 12,785 to 384,877 IU/mL (median 139,593 IU/mL; IQR 66,923–281,914). Five of six patients (83%) started maribavir with CMV DNA $\geq 50,000$ IU/mL. Because only one patient was below this threshold, no subgroup comparison was undertaken and no differential conclusion can be drawn from this distribution. Maribavir was selected in the setting of suboptimal virological control under prior anti-CMV therapy and/or concerns about toxicity.

All patients received oral maribavir 400 mg twice daily. Treatment duration ranged from 23 to 56 days (median 43.5 days). Four patients received a short adjunctive intervention during the first three maribavir days (foscarnet and/or anti-CMV immunoglobulin) as early intensification (Table 1).

Table 1. Patient characteristics and outcomes.

Patient	Age/Sex	Transplant (D/R)	Baseline CMV DNA (IU/mL)	Adjunctive Therapy (First 3 Days)	MBV * Duration (Days)	Outcome
1	24/M	Kidney transplant (SOT) (D+/R−)	384,877	Foscarnet	28	EOT: ↓ to <1000 IU/mL (not <LLOQ); Early rebound: No; AEs: None
2	39/F	Kidney transplant (SOT) (D+/R−)	192,754	Anti-CMV immunoglobulin	23	EOT: ↓ to <1000 IU/mL (not <LLOQ); Early rebound: No; AEs: None
3	53/F	Kidney transplant (SOT) (D+/R−)	86,432	Foscarnet	40	EOT: CMV DNA < 1000 IU/mL (not <LLOQ); documented rebound: No; AEs: None
4	49/F	Kidney transplant (SOT) (D+/R−)	311,634	None	49	EOT: CMV DNA < 1000 IU/mL (not <LLOQ); rebound: day 21; AEs: None
5	50/F	Kidney transplant (SOT) (D+/R−)	60,420	Anti-CMV immunoglobulin	56	EOT: CMV DNA < 1000 IU/mL (not <LLOQ); rebound: day 15; AEs: None
6	59/F	Autologous HSCT (R+)	12,785	None	47	EOT: confirmed clearance (<LLOQ on two consecutive tests); rebound: day 18 with recurrent fatigue; AEs: dysgeusia and mild fatigue

* Abbreviations: AE(s), adverse event(s); D/R, donor/recipient CMV serostatus; D+, donor seropositive; EOT, end of treatment; HSCT, hematopoietic stem cell transplantation; LLOQ, lower limit of quantification; MBV, maribavir; R-, recipient seronegative; R+, recipient seropositive; SOT, solid organ transplantation; ↓, reduction but not clearance.

Maribavir was associated with substantial reductions in CMV DNAemia. At the end of treatment, Patients 1–5 had detectable CMV DNA < 1000 IU/mL but did not meet the definition of confirmed clearance. Patient 6 was the only patient to achieve confirmed clearance (<LLOQ on two consecutive tests); this patient subsequently experienced virological and clinical rebound 18 days after discontinuation.

Brief case summaries are provided below. Post-treatment observation time, prior therapy, and retreatment details should be reported in Table 1.

Patient 1 (kidney transplant) initiated maribavir at 384,877 IU/mL, received brief foscarnet bridging, ended treatment with CMV DNA < 1000 IU/mL (not <LLOQ), and had no documented rebound during days after discontinuation.

Patient 2 (kidney transplant) initiated maribavir at 192,754 IU/mL with adjunctive anti-CMV immunoglobulin, ended treatment with CMV DNA < 1000 IU/mL (not <LLOQ), and had no documented rebound during days after discontinuation.

Patient 3 (kidney transplant) initiated maribavir at 86,432 IU/mL, received foscarnet bridging, completed 40 days of maribavir, ended treatment with CMV DNA < 1000 IU/mL (not <LLOQ), and had no documented rebound during days after discontinuation.

Patient 4 (kidney transplant) initiated maribavir at 311,634 IU/mL and experienced virological rebound 21 days after discontinuation, without CMV-attributable clinical symptoms. Retreatment, genotyping at rebound, response to retreatment, and final outcome should be reported in Table 1.

Patient 5 (kidney transplant) initiated maribavir at 60,420 IU/mL, completed 56 days of treatment, and experienced virological rebound 15 days after discontinuation, without CMV-attributable clinical symptoms. Retreatment, genotyping at rebound, response to retreatment, and final outcome should be reported in Table 1.

Patient 6 (autologous HSCT) initiated maribavir at 12,785 IU/mL, completed 47 days of treatment, achieved confirmed clearance (<LLOQ on two consecutive tests), and experienced virological and clinical rebound 18 days after discontinuation, with recurrent fatigue. Retreatment, genotyping at rebound, response to retreatment, and final outcome should be reported in Table 1.

Early post-treatment rebound requiring additional anti-CMV therapy occurred in 3/6 patients within 15–21 days of discontinuation. The other three patients had no documented rebound, but absence of rebound is interpretable only in relation to the duration of post-treatment observation, which should be reported for each patient. Rebound occurred in two SOT recipients with baseline CMV DNA $\geq 50,000$ IU/mL and in the HSCT recipient with a lower baseline viral load; the sample is insufficient for comparison by baseline viral-load category.

Maribavir was well tolerated. One patient reported dysgeusia and mild fatigue; no patient discontinued maribavir due to adverse events. No clinically meaningful nephrotoxicity or cytopenias attributable to maribavir were observed.

4. Discussion

In this single-center case series, maribavir was well tolerated and was associated with substantial reductions in CMV DNAemia in patients with refractory CMV infection. Three of six patients experienced early post-treatment rebound within 15–21 days, including Patient 6, the only patient who achieved confirmed clearance; one rebound was accompanied by recurrent fatigue. These findings identify uncertainty around treatment endpoints but, given the sample size and retrospective design, cannot establish a stopping rule or management algorithm [13–16].

Residual low-level DNAemia at the end of treatment was followed by rebound in two patients and no documented rebound in three, whereas confirmed clearance was followed by rebound in the single patient who achieved it. Thus, neither residual DNAemia nor confirmed clearance can be considered a reliable predictor of durability on the basis of this cohort. The observations should be interpreted as hypothesis-generating rather than as evidence for a particular stopping strategy.

Clinical improvement and a marked decline in CMV DNAemia remain meaningful outcomes in refractory CMV infection, but the ultimate goal is durable viral control without relapse. In the SOLSTICE trial, maribavir improved confirmed viremia clearance at week 8 compared with investigator-assigned therapy, although many patients did not achieve formal clearance [5]. Our observations illustrate the gap between short-term viral reduction and durable post-treatment control, but they do not identify the mechanism of rebound or a validated surrogate endpoint.

In our cohort, three of six patients rebounded within 15–21 days after maribavir discontinuation and required additional anti-CMV therapy. The early timing suggests that the first weeks after discontinuation are an important period for observation; however, this series cannot validate a specific testing interval or duration. Post-treatment follow-up should be interpreted in the context of per-patient observation time, viral kinetics, intensity of immunosuppression, organ involvement, and immune recovery. Any proposed schedule requires prospective evaluation.

This study cannot determine whether maribavir should be stopped at a low-level plateau, continued for longer, or replaced with another antiviral. Extending treatment might permit further viral decline in some patients, but it might also prolong drug exposure without additional benefit. Conversely, discontinuation may be followed by early rebound even after confirmed clearance, as occurred in Patient 6. Therefore, the present findings should not be interpreted as clinical guidance for stopping maribavir.

Because systematic genotyping was not performed, rebound cannot be attributed to maribavir resistance. Alternative explanations include incomplete CMV-specific immune recovery, continued immunosuppression, residual replication, variability in adherence or antiviral exposure, and assay variability near the LLOQ. Resistance remains one theoretical possibility, but the present data cannot distinguish among these mechanisms and no resistance-related conclusion should be drawn without documented per-patient UL97/UL54 results.

Baseline viral load is relevant context because prescribing information reports lower response rates when baseline CMV DNA is $\geq 50,000$ IU/mL [6]. In this cohort, five of six patients were above that threshold and only one was below it; therefore, no comparison between viral-load subgroups is possible. Four patients received short adjunctive treatment with foscarnet and/or anti-CMV immunoglobulin during the first three maribavir days. This heterogeneous, non-randomized use cannot establish the efficacy of bridging, adjunctive, or sequential strategies, and the series should not be used to recommend a specific treatment sequence [8,17–22].

With regard to safety, maribavir was well tolerated, in line with clinical trial data [23–26]. Only mild dysgeusia and fatigue were recorded, and no patient discontinued treatment because of toxicity [5,6]. The small sample does not permit comparative safety conclusions, but no clinically meaningful nephrotoxicity or cytopenias attributable to maribavir were observed.

This study is limited by its small sample size, retrospective design, heterogeneous management, and incomplete resistance testing. CMV-specific immunity was not assessed, and changes in immunosuppression were not analyzed systematically. Recovery of CMV-specific immune function may be an important determinant of durable control and could help explain why similar virological endpoints were followed by different outcomes. Complete reporting of follow-up duration, prior antiviral exposure, retreatment, and final outcome is also necessary for interpretation.

Future multicenter studies should combine standardized longitudinal viral kinetics, CMV-specific immune assessment, systematic genotyping, antiviral-exposure data, and prespecified follow-up to evaluate treatment duration, stopping criteria, and relapse risk. This case series primarily identifies variables and endpoints that merit prospective study rather than providing practice-changing recommendations.

5. Conclusions

In this six-patient real-world case series, maribavir was well tolerated and was associated with substantial CMV DNA reductions in transplant recipients with refractory CMV. Early rebound occurred after discontinuation in three patients, including the only patient with confirmed clearance. The findings do not establish that stopping at detectable DNAemia is safe, that confirmed clearance prevents relapse, or that a specific monitoring or sequential-treatment strategy is optimal.

These observations should be considered hypothesis-generating. Prospective studies with complete longitudinal viral kinetics, standardized follow-up, CMV-specific immune assessment, and systematic genotyping are needed to define treatment duration, stopping criteria, and relapse risk after maribavir discontinuation.

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Data Availability Statement: The raw data supporting the conclusions of this article will be made available by the authors on reasonable request.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

CMV	Cytomegalovirus
R/R CMV	Refractory/resistant cytomegalovirus (general literature term)
SOT	Solid organ transplantation
HSCT	Hematopoietic stem cell transplantation
LLOQ	Lower limit of quantification
MBV	Maribavir
EOT	End of treatment
AE	Adverse event
D/R	Donor/recipient CMV serostatus

References

1. Ljungman, P.; Hirsch, H.H.; Chemaly, R.F.; Khawaya, F.; Alain, S.; Avery, R.; Badshah, C.; Boeckh, M.; Fournier, M.; Hodowanec, A.; et al. Consensus Definitions of Cytomegalovirus (CMV) Infection and Disease in Transplant Patients Including Resistant and Refractory CMV for Use in Clinical Trials: 2024 Update from the Transplant-Associated Virus Infections Forum. *Clin. Infect. Dis.* **2024**, *79*, 787–794. [[CrossRef](#)]
2. Stewart, C.L.; Kotton, C.N. What’s New: Updates on Cytomegalovirus in Solid Organ Transplantation. *Transplantation* **2024**, *108*, 884–897. [[CrossRef](#)] [[PubMed](#)]
3. Khawaja, F.; Zamora, D.; Yong, M.K.; Hakki, M.; Goscicki, B.K.; Danziger-Isakov, L.; Lin, A.; Carpenter, P.A.; Boeckh, M.; Papanicolaou, G.A.; et al. American Society for Transplantation and Cellular Therapy Series #11: Updated Cytomegalovirus Guidelines in Hematopoietic Cell Transplant and Cellular Therapy Recipients. *Transplant. Cell. Ther.* **2025**, *31*, 727–741. [[CrossRef](#)] [[PubMed](#)]
4. Girmenia, C.; Lazzarotto, T.; Martino, M.; Bonifazi, F.; Baldanti, F.; Clerici, P.; Citterio, F.; De Carlis, L.; Barosi, G.; Grossi, P.A. Management of Cytomegalovirus Infection in Allogeneic Hematopoietic Stem Cell and in Solid Organ Transplantation: Updated Recommendations by the GITMO, SITO, SIMIT, and AMCLI Italian Societies. *Clin. Transplant.* **2025**, *39*, e70255. [[CrossRef](#)] [[PubMed](#)]
5. Avery, R.K.; Alain, S.; Alexander, B.D.; Blumberg, E.A.; Chemaly, R.F.; Cordonnier, C.; Duarte, R.F.; Florescu, D.F.; Kamar, N.; Kumar, D.; et al. Maribavir for Refractory Cytomegalovirus Infections with or without Resistance Post-Transplant: A Phase 3 Randomized Clinical Trial. *Clin. Infect. Dis.* **2022**, *75*, 690–701. [[CrossRef](#)] [[PubMed](#)]
6. Takeda Pharmaceuticals U.S.A. Inc. *LIVTENCITY (Maribavir) Tablets: U.S. Prescribing Information*; Revised November; Takeda Pharmaceuticals U.S.A., Inc.: Cambridge, MA, USA, 2025.
7. European Medicines Agency. *Livtency (Maribavir): EPAR—Product Information*; European Medicines Agency (EMA): Amsterdam, The Netherlands, 2022.
8. Ogawa, L.; Shum, S.; Mitra, S.; Gaynor, P.; Beaird, O.E.; Pham, C.; Schaenman, J.M. Real-World Comparison of Maribavir to Foscarnet for the Treatment of Cytomegalovirus in Solid Organ and Hematopoietic Stem Cell Transplant Recipients. *Viruses* **2024**, *16*, 1889. [[CrossRef](#)] [[PubMed](#)]
9. Chou, S.; Mouas, H.; Cervera, C.; Chemaly, R.F.; Kotton, C.N.; Lundgren, J.; Papanicolaou, G.A.; Pereira, M.R.; Wu, J.J.; Murray, R.A.; et al. Drug Resistance Assessed in a Phase 3 Clinical Trial of Maribavir Therapy for Refractory or Resistant Cytomegalovirus Infection in Transplant Recipients. *J. Infect. Dis.* **2024**, *229*, 413–421. [[CrossRef](#)] [[PubMed](#)]

10. Papanicolaou, G.A.; Avery, R.K.; Cordonnier, C.; Duarte, R.F.; Haider, S.; Maertens, J.; Peggs, K.S.; Solano, C.; Young, J.-A.H.; Fournier, M.; et al. Treatment for First Cytomegalovirus Infection Post–Hematopoietic Cell Transplant in the AURORA Trial: A Multicenter, Double-Blind, Randomized, Phase 3 Trial Comparing Maribavir with Valganciclovir. *Clin. Infect. Dis.* **2024**, *78*, 562–572. [[CrossRef](#)] [[PubMed](#)]
11. Chou, S.; Alain, S.; Winston, D.J.; Cervera, C.; Duarte, R.F.; Haider, S.; Maertens, J.; Peggs, K.S.; Solano, C.; Young, J.-A.H.; et al. Comparative Emergence of Maribavir and Ganciclovir Resistance in a Randomized Phase 3 Clinical Trial for Treatment of Cytomegalovirus Infection. *J. Infect. Dis.* **2025**, *231*, e470–e477. [[CrossRef](#)] [[PubMed](#)]
12. Watanabe, K.; Chou, S. Ganciclovir and Maribavir Cross-Resistance Revisited. *Antivir. Res.* **2023**, *222*, 105792. [[CrossRef](#)] [[PubMed](#)]
13. Razonable, R.R. Current Perspectives on Letermovir and Maribavir for the Management of Cytomegalovirus Infection in Solid Organ Transplant Recipients. *Drug Des. Devel Ther.* **2024**, *18*, 3987–4001. [[CrossRef](#)] [[PubMed](#)]
14. Halpern-Cohen, V.; Blumberg, E.A. New Perspectives on Antimicrobial Agents: Maribavir. *Antimicrob. Agents Chemother.* **2022**, *66*, e0240521. [[CrossRef](#)] [[PubMed](#)]
15. Heldman, M.R.; Limaye, A.P. Maribavir versus investigator assigned therapy: Exploring the exploratory. *Transpl. Infect. Dis.* **2023**, *25*, e14063. [[CrossRef](#)] [[PubMed](#)]
16. Kleiboeker, H.L.; Descourouez, J.L.; Schulz, L.T.; Mandelbrot, D.A.; Odorico, J.S.; Rice, J.P.; Saddler, C.M.; Smith, J.A.; Jorgenson, M.R. Maribavir for the Management of Cytomegalovirus in Adult Transplant Recipients: A Review of the Literature and Practical Considerations. *Ann. Pharmacother.* **2023**, *57*, 597–608. [[CrossRef](#)] [[PubMed](#)]
17. Walti, C.S.; Khanna, N.; Avery, R.K.; Helanterä, I. New Treatment Options for Refractory/Resistant CMV Infection. *Transpl. Int.* **2023**, *36*, 11785. [[CrossRef](#)] [[PubMed](#)]
18. Gourin, C.; Alain, S.; Hantz, S. Anti-CMV therapy, what next? A systematic review. *Front. Microbiol.* **2023**, *14*, 1321116. [[CrossRef](#)] [[PubMed](#)]
19. El Helou, G.; Razonable, R.R. Safety considerations with current and emerging antiviral therapies for cytomegalovirus infection in transplantation. *Expert Opin. Drug Saf.* **2019**, *18*, 1017–1030. [[CrossRef](#)] [[PubMed](#)]
20. Danziger-Isakov, L.; Mark Baillie, G. Hematologic complications of anti-CMV therapy in solid organ transplant recipients. *Clin. Transplant.* **2009**, *23*, 295–304. [[CrossRef](#)] [[PubMed](#)]
21. Charlotte, R.; François, P.; Jonathan, M.; Véronique, B.; Olivier, B.; Tristan, D.; Séverine, F.; Jérôme, L.; Adrien, T.; Claire, D.; et al. Use of anti-CMV immunoglobulins in lung transplant recipients: The French experience. *Transpl. Infect. Dis.* **2021**, *23*, e13754. [[CrossRef](#)] [[PubMed](#)]
22. Khare, M.D.; Sharland, M. Cytomegalovirus treatment options in immunocompromised patients. *Expert Opin. Pharmacother.* **2001**, *2*, 1247–1257. [[CrossRef](#)] [[PubMed](#)]
23. Gandhi, R.G.; Kotton, C.N. Evaluating the Safety of Maribavir for the Treatment of Cytomegalovirus. *Ther. Clin. Risk Manag.* **2022**, *18*, 223–232. [[CrossRef](#)] [[PubMed](#)]
24. Song, I.; Chen, G.; Wu, J.; Ilic, K. Maribavir Pharmacokinetics and Safety in Participants with Moderate Hepatic Impairment: A Phase 1, Open-Label, Single-Dose, Parallel Group Study. *J. Clin. Pharmacol.* **2023**, *63*, 250–258. [[CrossRef](#)] [[PubMed](#)]
25. Song, I.; Suttle, B.; Wu, J.; Ilic, K. Pharmacokinetics and Safety Evaluation of Maribavir in Healthy Japanese and Matched White Participants: A Phase 1, Open-Label Study. *Clin. Pharmacol. Drug Dev.* **2023**, *12*, 645–654. [[CrossRef](#)] [[PubMed](#)]
26. Gong, Q.; Zhang, L.; Wu, H.; Miao, Y.; Huang, L.; Yin, C.; Hu, Y. Real-world safety of maribavir: A retrospective study based on signal detection in the FDA adverse event reporting system. *Int. J. Clin. Pharm.* **2025**, *47*, 767–774. [[CrossRef](#)] [[PubMed](#)]

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