Valganciclovir: An Advance in Cytomegalovirus Therapeutics

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OBJECTIVE: To review the pharmacology, pharmacokinetics, and preliminary clinical data for valganciclovir, a new oral agent for the therapy of cytomegalovirus (CMV) retinitis.

DATA SOURCES: Relevant literature was extracted via MEDLINE/PUBMED and searchable abstracts from infectious diseases conferences covering the period from January 1990 to April 2002. Tertiary references provided background information.

DATA SYNTHESIS: Current standard treatment for CMV retinitis consists of intravenous therapy, intraocular implant, and intraocular injection. The low bioavailability of oral ganciclovir restricts its use to prophylaxis and maintenance treatment. Oral valganciclovir, recently approved for both induction and maintenance therapy of CMV retinitis, may fill a niche for this disease.

CONCLUSIONS: Although only 1 clinical study has been published for valganciclovir, its favorable pharmacokinetic profile, encouraging preliminary efficacy data, ease of administration, and lack of potential catheter-related complications make it a favorable option for the treatment of CMV retinitis in HIV-positive patients.

KEY WORDS: cytomegalovirus retinitis, ganciclovir, valganciclovir.

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Cytomegalovirus (CMV) is a common pathogen in immunocompromised hosts. The virus may affect many different body systems including the central nervous system, respiratory tract, and gastrointestinal tract. In patients with AIDS, CMV most commonly manifests itself as necrotizing retinitis.[1,2] Untreated disease can progress to retinal scarring, retinal detachment, loss of vision, and, ultimately, blindness.[2] Although incidence has declined greatly due to the advent of highly active antiretroviral therapy (HAART), CMV retinitis remains an important opportunistic infection due to the great impact it can have on a patient's quality of life.

Effective treatments for CMV retinitis remain limited. Utilizing HAART to treat the underlying HIV infection may aid in controlling CMV retinitis if immune reconstitution occurs. Unfortunately, not all patients will experience CD4+ counts sufficiently high enough or sustained long enough to provide adequate protection against CMV disease.[3] Patients will require induction therapy for 14–21 days to control their retinitis, then usually lifetime maintenance therapy. Intravenous ganciclovir remains the drug of choice for induction and maintenance due to its effectiveness and tolerability. For those that fail intravenous ganciclovir, foscarnet and cidofovir are alternative therapeutic options. All 3 agents are effective for induction and maintenance therapy of CMV retinitis.[4] Intravenous therapy with these medications poses the risk of complications from catheter-related complications such as thrombophlebitis, line infection, and bacteremia.[5] Foscarnet treatment is associated with nephrotoxicity and electrolyte abnormalities such as hypocalcemia, hypomagnesemia, hypo/hyperphosphatemia, and hypokalemia.[6] Cidofovir treatment is also associated with nephrotoxicity, hypotony, and rash (secondary to concomitant probenecid).[7] These unfavorable adverse effects have caused these 2 drugs to be considered second-line alternatives.

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An oral drug for CMV retinitis could potentially reduce patient inconvenience, nursing time, and potential morbidity due to therapy complications. Oral ganciclovir capsules are currently available on the market. Unfortunately, these capsules exhibit a low bioavailability of 5–9% and are only approved for prophylaxis and maintenance therapy for CMV retinitis.[8] Furthermore, the regimen is inconvenient (two 500-mg capsules 3 times daily), expensive, and of questionable clinical benefit.

Until recently, patients newly diagnosed with this disease required intravenous therapy for the duration of the induction phase, if not also for maintenance. In March 2001, the Food and Drug Administration (FDA) approved valganciclovir, a new oral drug for the induction and maintenance therapy of CMV retinitis. It is the objective of this article to review valganciclovir pharmacology, pharmacokinetics, available clinical information, and economic considerations.

Data Sources

A MEDLINE/PUBMED search (January 1990–April 2002) was performed to identify articles containing the key word valganciclovir. Abstracts from the International AIDS Conference, Conference on Retroviruses and Opportunistic Infections, Interscience Conference on Antimicrobial Agents and Chemotherapy, and the International AIDS Society Conference on HIV Pathogenesis and Treatment were also included in this literature search. It was necessary to also include the information from unpublished clinical trials from the product package insert.[9]

Pharmacology

Valganciclovir is an L-valine ester prodrug of ganciclovir. After ingestion, valganciclovir is transported from the intestine to the bloodstream by the peptide transporter PEPT1.[10] Liver and intestinal esterases rapidly hydrolyze valganciclovir to its active form, ganciclovir. Ganciclovir is a 2′-deoxyguanosine (purine) nucleoside analog that is converted in the body to ganciclovir monophosphate by phosphotransferase, the UL97 gene product of the CMV virus.[4] The compound is further modified in the body by other phosphorylases.[11] The final active compound, ganciclovir triphosphate, is then incorporated at the 3′ site of growing viral DNA.[4,12] The ganciclovir-modified strand is conformationally less stable due to the acyclic sugar side chain. As a consequence, elongation of that strand is severely delayed. Resistance to ganciclovir has been reported[4] via mutations in the pUL97 phosphotransferase and UL54 viral polymerase genes, leading to reduced incorporation of ganciclovir into the DNA strand.

Pharmacokinetics and Pharmacodynamics

Two published studies have explored the pharmacokinetic properties of valganciclovir. Their results are summarized in Table 1.[9,13,14] In a randomized, open-label, crossover study,[13] 18 HIV- and CMV-positive individuals without active CMV disease were randomized to receive either a single dose of valganciclovir 360 mg, a single oral dose of ganciclovir 1000 mg, or intravenous ganciclovir 5 mg/kg over 1 hour. A 1-week washout period was used between each course of therapy. Study results indicated that valganciclovir was rapidly converted to ganciclovir, with a mean plasma half-life of 0.47 ± 0.18 hours. In addition, the absolute bioavailability of valganciclovir was 60.9% versus 5.6% for oral ganciclovir. The mean ± SD ganciclovir AUC after valganciclovir administration was 10.8 ± 1.9 µg•h/mL versus an intravenous ganciclovir AUC of 25.1 ± 3.8 µg•h/mL. The AUC was <50% of that of a 5-mg/kg dose of intravenous ganciclovir. Despite being a single-dose study, this trial provided preliminary pharmacokinetic data that suggested approximately doubling the valganciclovir dose could approximate the usual 5-mg/kg intravenous ganciclovir dose used for CMV therapy.

In an open-label, randomized, crossover, dose-ranging study,[14] 39 HIV- and CMV-negative volunteers received valganciclovir 450, 875, 1750, or 2625 mg once daily for 3 days with a 4-day washout period between each dosing regimen. Thirty-two individuals completed this multiple-dose pharmacokinetic study. At the 875-mg dose administered with food, the maximum concentration was 6.07 mg/L with an AUC_{24} of 24.8 mg/L•h. Based on the data, the authors suggest that a 900-mg dose would approximate the target AUC_{24} value of 26 mg/L•h achieved with intravenous ganciclovir 5 mg/kg.

Table 1. Pharmacokinetic Parameters for Ganciclovir After Administration of Valganciclovir

Parameter	Jung and Dorr (1999)[13] Fasted State 360 mg/d	Brown et al. (1999)[14] Fasted State 450 mg/d	Brown et al. (1999)[14] Fasted State 2625 mg/d	Brown et al. (1999)[14] Fed State 450 mg/d	Brown et al. (1999)[14] Fed State 2625 mg/d	Package Insert[9] Fed State 900 mg/d
Bioavailability (%)	60.9 ± 9.1					59.4 ± 6.1
t_{max} (h)	1.03 ± 0.34	1.0	1.75	1.5	2.0	
C_{max} (µg/mL)	2.98 ± 0.77	3.1	12.3	3.28	15.4	5.61 ± 1.52
AUC (µg•h/mL)	10.8 ± 1.9	10.3	47.3	12.7	74.1	29.1 ± 9.7
CL (mL/min/kg)						3.21 ± 0.75
$t_{1/2}$ (h)	3.69 ± 0.62	3.92	4.54	3.80	4.42	4.08 ± 0.76

CL = clearance; C_{max} = maximum concentration; $t_{1/2}$ = terminal half-life; t_{max} = time to reach maximum concentration.

The study also noted a statistically significant increase in bioavailability when valganciclovir was dosed in the fed versus fasted state. At the 875-mg dose, the mean valganciclovir AUC_{24} (fasted) was 0.328 mg/L•h versus AUC_{24} (fed) 0.393 mg/L•h. At the 875-mg dose, the mean ganciclovir AUC_{24} (fasted) was 19.0 mg/L•h versus AUC_{24} (fed) 24.8 mg/L•h. Between the fed and fasted states, the relative bioavailability increased 30% (95% CI 1.21 to 1.51; $p < 0.001$). Although the authors did not employ a control or comparator (intravenous ganciclovir) group, the trial provided valuable pharmacokinetic data on the need for food to ensure adequate absorption and the approximate oral dose that would provide comparable efficacy to intravenous ganciclovir.

Clinical Trials

Only 1 peer-reviewed clinical trial has been published regarding valganciclovir efficacy for induction therapy. Martin et al.[15] compared valganciclovir 900 mg with intravenous ganciclovir 5 mg/kg. This randomized, controlled clinical trial enrolled 160 HIV-positive patients newly diagnosed with CMV retinitis for a 21-day induction course followed by maintenance therapy. Valganciclovir was initiated at 900 mg twice daily for 21 days, followed by 900 mg daily. The primary endpoint for the study was progression of CMV retinitis within 4 weeks of treatment initiation. Approximately 10% of patients in each treatment arm exhibited progression of their retinitis after 4 weeks (10% vs. 9.9%; 95% CI −0.097 to 0.100). Seventy-seven percent of patients (n = 47) had a satisfactory response to intravenous ganciclovir induction therapy compared with 72% of patients (n = 46) receiving valganciclovir (95% CI −0.204 to 0.101). The median time to first progression was 160 days and 125 days for valganciclovir and ganciclovir, respectively. Mean ± SD AUCs were similar between both treatment arms for induction (28.6 ± 9.0 vs. 32.8 ± 10.1 mg•h/mL for intravenous ganciclovir and valganciclovir, respectively). For maintenance, the mean AUCs were 30.7 ± 7.7 versus 34.9 ± 13.3 mg•h/mL for intravenous ganciclovir and valganciclovir, respectively. Valganciclovir bioavailability was determined to be 60%. Rates of adverse events were similar between the 2 treatment groups. Although the higher AUC and longer time to first progression appear to favor valganciclovir, the study was designed to prove noninferiority, and it probably does not represent a significant difference.

No clinical trials have been published regarding comparative efficacy of valganciclovir for maintenance therapy of CMV retinitis, although clinical trial data listed in the product package insert[9] included valganciclovir maintenance therapy as part of the open-label portion of the study, as noted previously. Rates of ganciclovir resistance during valganciclovir therapy appear to be comparable to intravenous ganciclovir. After a median therapy duration of 182 days, 9.4% (14/148) of patients had evidence of genotypic resistance to ganciclovir (UL97 mutations).[16]

Adverse Effects and Drug Interactions

Common adverse effects for valganciclovir are summarized in Table 2. Vomiting, abdominal pain, fever, thrombocytopenia, insomnia, peripheral neuropathy, paresthesias, and retinal detachment have also been reported.[9] Drug interactions for valganciclovir are expected to be similar to those for ganciclovir since the prodrug-to-active drug conversion is rapid. Consequently, no drug–drug interaction studies have been conducted; the package insert lists only drug–drug interactions that have been obtained from the ganciclovir package insert. Among these, clinically significant interactions include zidovudine (increased risk for bone marrow suppression such as anemia and neutropenia) and didanosine (increased risk of pancreatitis or peripheral neuropathy secondary to increased didanosine absorption). Patients with renal impairment taking both mycophenolate mofetil and valganciclovir should be monitored for additive toxicity. Probenecid blocks the renal tubular secretion of ganciclovir, increasing ganciclovir concentrations, possibly resulting in greater risk of bone marrow suppression.

Use in Special Populations

There are no clinical studies supporting the use of valganciclovir in children and the elderly. Careful consideration should be observed before starting these patients on valganciclovir since medications tend to have different pharmacodynamic and pharmacokinetic properties in these populations. There are no studies supporting the use of valganciclovir in pregnant or lactating women. It is anticipated that valganciclovir would be harmful to the growing fetus, similar to ganciclovir. Based on this, the FDA has labeled valganciclovir as a pregnancy category C (animal studies show harmful effects on the fetus; no data in humans).

Patients with impaired renal function will require dosage reduction for their valganciclovir regimen.[9] Patients with creatinine clearances between 40 to 59 mL/min should receive half of the standard induction dose and half of the standard maintenance dose (450 mg twice daily and 450 mg once daily, respectively). Patients with creatinine clearances between 25

Table 2. Selected Adverse Effects[9]

Adverse Event[a]	Valganciclovir[b]	Intravenous Ganciclovir[b]	Valganciclovir[c]
Anemia	8	8	26
Catheter-related infections	3	11	not available
Diarrhea	16	10	41
Headache	9	5	22
Nausea	8	14	30
Neutropenia	11	13	27

[a]Data presented as percentages.
[b]Study WV15376 (unpublished, n = 79 pts./arm).
[c]Pooled study data from 2 unpublished clinical trials (n = 370).

and 39 mL/min should receive 450 mg/d for induction and 450 mg every other day for maintenance. Patients with creatinine clearances of 10–24 mL/min should receive 450 mg every other day for induction and 450 mg twice weekly for maintenance. Patients on hemodialysis are unfavorable candidates for valganciclovir therapy since they require dosage reductions too impractical to achieve with the 450-mg valganciclovir tablets.

Therapeutic and Economic Issues

The early, favorable results from pharmacokinetic and clinical studies likely led to FDA approval of valganciclovir for the induction and maintenance treatment of CMV retinitis. Recently published and other unpublished studies suggest similar efficacy to intravenous ganciclovir therapy.

This oral therapy for CMV would appeal to patients primarily due to its convenience compared with intravenous therapy. The regimen is uncomplicated, with its twice-daily or once-daily dosing schedule, theoretically improving patient acceptance and adherence without the risk of intravenous line complications. However, valganciclovir is not a completely benign drug, and patients will have to be monitored as frequently as their counterparts receiving intravenous therapy.

A cost comparison of valganciclovir and ganciclovir is provided in Table 3.[17] When viewing these data, administration-associated costs such as nursing time must also be taken into consideration. The oral dosage form of valganciclovir may lead to a potential reduction in institutional costs. This potential reduction is multifactorial. Patients would no longer be required to come to the hospital or infusion center to receive ganciclovir infusions, decreasing staff time required for administration and monitoring. Similarly, valganciclovir would also decrease the need for home-health nursing visits. Ideally, valganciclovir tablets would decrease the incidence of infusion-related complications, saving the institution the cost of managing these adverse events.

Approved Labeling and Possible Off-Label Uses

Valganciclovir is indicated for both induction and maintenance therapy for CMV retinitis in HIV-positive patients.

Table 3. Cost of Therapy Based on Average Wholesale Price[17]

Parameter	Induction ($)	Maintenance ($)
Intravenous ganciclovir	3084.00[a]	1542.00[b]
Oral ganciclovir		1664.92[c]
Valganciclovir	1611.46[d]	1726.56[e]

[a]Based on 5 mg/kg every 12 h for 2 wk in a 70-kg pt.
[b]Based on 5 mg/kg/d, 5 d each wk, for 4 wk.
[c]Based on two 500-mg capsules 3 times daily for 30 d (pricing is same for 250-mg capsules).
[d]Based on 2 tablets twice daily for 2 wk.
[e]Based on 2 tablets daily for 30 d.

It is being investigated for the prophylaxis or treatment of CMV infection in solid organ and bone marrow transplant patients.

Dosage and Administration

Valganciclovir is supplied as a bottle of sixty 450-mg pink tablets with the letters VGC and 450 imprinted on either side. The medication should be stored between 15 and 30 ºC. Tablets should not be broken or crushed for risk of exposure to a potential carcinogen. Valganciclovir tablets cannot be substituted for ganciclovir capsules on a one-to-one basis.[16]

Patient Counseling

Patients should be advised to take valganciclovir at the same time each day, with food. A calendar may be useful to keep track of when to switch from twice-daily dosing (induction therapy) to once-daily dosing (maintenance therapy). Regardless of gender, patients should use appropriate contraception and should speak with their providers if they are considering having children. Women taking valganciclovir should not breast-feed. Common adverse effects such as diarrhea, nausea, fever, and headache should be reviewed with the patient. Patients should also be encouraged to discuss any unusual adverse effects with their providers. Adherence to the scheduled appointments for laboratory tests also should be stressed.

Formulary Recommendations and Summary

Given the kinetics and available information, valganciclovir appears to be comparable to intravenous ganciclovir without the risks of catheter infection and inconvenience of daily infusions. Institutions may benefit from a potential decrease in overall direct costs due to decreased use of infusion bed time, elimination of administration/preparation time and its associated costs, reduced catheter infections, and increased patient adherence. The above factors could potentially make valganciclovir a cost-effective option for patients needing CMV induction or maintenance therapy.

Patient selection for valganciclovir therapy would include those with a proven "track record" of adherence to provider and laboratory appointments and have non-Zone-1 disease. Patients presenting with Zone 1 disease requiring induction therapy probably should receive at least several days of intravenous ganciclovir induction, if not a complete induction, before switching to valganciclovir as valganciclovir has not been studied in patients with Zone 1 disease. Appropriate monitoring parameters would include a complete blood cell count with differential and serum creatinine 2–3 times weekly during the induction phase, then weekly or twice weekly during maintenance therapy. Frequency of monitoring can be reduced to every 2 weeks during maintenance therapy. Before starting or switching to valganciclovir, a baseline ophthalmologic examination should be obtained, allowing the provider to easily estab-

lish healing or progression of CMV disease while on oral therapy. If CMV retinitis continues to progress despite valganciclovir therapy, and malabsorption, subtherapeutic dosing, or nonadherence can be eliminated as a cause, the virus may have developed resistance to ganciclovir. In such a case, alternative therapy is required.

Valganciclovir remains an important advance in CMV therapy despite the relative lack of data. Considerably more information on its efficacy and pharmacoeconomics is needed. To this end, the authors strive to collect data on valganciclovir effectiveness, tolerability, and economics in actual clinical practice.

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EXTRACTO

OBJETIVO: Evaluar la farmacología, farmacocinética, y datos clínicos preliminares del valganciclovir, un agente oral nuevo para el tratamiento de retinitis por cytomegalovirus (CMV).

FUENTES DE DATOS: Se utilizó literatura relevante, extraída utilizando la base de datos de MEDLINE/PUBMED y extractos de conferencias de infectología para el período comprendido entre enero de 1990 a diciembre de 2001. Se utilizaron referencias terciarias para información de trasfondo.

SÍNTESIS DE DATOS: El tratamiento estándar de retinitis por cytomegalovirus consiste de terapia endovenosa, implantes intraoculares, e inyecciones intraoculares. La pobre biodisponibilidad de ganciclovir administrado por la vía oral limita su uso para la profilaxis y terapia de mantenimiento. Valganciclovir oral ha sido aprobado recientemente para la inducción y terapia de mantenimiento de retinitis por cytomegalovirus.

CONCLUSIONES: Aunque no se han publicado estudios clínicos para valganciclovir, su perfil farmacocinético favorable, datos preliminares de eficacia prometedores, la facilidad de la administración, y la ausencia de complicaciones asociados al catéter hacen esta modalidad de tratamiento una opción favorable para el tratamiento de retinitis por CMV en pacientes infectados con el Virus de Inmunodeficiencia Humana.

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RÉSUMÉ

OBJECTIF: Réviser la pharmacologie, la pharmacocinétique, et les données cliniques préliminaires du valganciclovir, un nouvel agent oral indiqué dans le traitement de la rétinite à cytomégalovirus (CMV).

SOURCES DE DONNÉES: Les articles pertinents ont été identifiés grâce à MEDLINE et des résumés de congrès d'infectiologie, entre janvier 1990 et décembre 2001. Des références tertiaires ont aussi été utilisées.

SYNTHÈSE: Le traitement standard de la rétinite à CMV consiste en des thérapies intraveineuses, des implants oculaires, et des injections intraoculaires. La faible biodisponibilité du ganciclovir oral limite son utilisation à des traitements prophylactiques ou de maintien. Le valganciclovir oral a récemment été approuvé pour l'induction et le traitement de la rétinite à CMV.

CONCLUSIONS: La pharmacocinétique favorable du valganciclovir, les données préliminaires encourageantes ainsi que la facilité d'administration permettent de croire qu'il deviendra une option thérapeutique favorable pour la traitement de la rétinite à CMV chez le patient infecté au VIH. Des études cliniques seront par contre nécessaires afin de positionner la molécule dans l'algorithme thérapeutique.

Jean Longtin