EXPLORING CYTOMEGALOVIRUS INFECTION IN RENAL TRANSPLANT RECIPIENTS: A COMPREHENSIVE LITERATURE REVIEW

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ARTICLE INFO

REVIEW

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Vol: 3 | Issue: 3
ISSN Print: 2960-2580
ISSN Online: 2960-2599

Publisher:
Medical Research and Statistical Consultancy Training Centre (SMC-PRIVATE) Limited

Muhammad Ajmal Dina: Drafted the manuscript
Asifa Karamat, Muhammad Bilal, Muhammad Shakeel Basit: Data management and critical appraisal
Muhmmad Akram Bhutta:
Zar Nigar, Shazia Siddique: Critical appraisal of the manuscript
Keywords:
Cytomegalovirus; kidney; Transplantation

ABSTRACT

Background: Complication in kidneys the cytomegalovirus (CMV) infection still important in transplantation, due to un favoring effects on grafts survival and general patient result, the role in energizing acute or chronic allograft injury, good recognize but the CMV linked nephritis is regularly undiagnosed, emphasizing about of timely heading and its management. **Objective**: The review aims to combine between current knowledge on CMV nephritis in the kidney transplant recipients emphasizing its fundamental mechanisms, diagnostic methods with therapeutic plans. **Methods**: This literature review was conducted counting 15 peer-reviewed articles published in 2010 to 2022, databases searched are PubMed, Google Scholar, Cochrane Library, and Mendeley, with the keywords of CMV kidney transplant" & cytomegalovirus nephritis." The data covered with patients' clinical presentation, risk factors, histopathology, demographics and outcomes. **Results**: The comprised studies were on transplant recipients aged rage between 38 to 62 years, representation of numerous ethnic groups and sexes showed with hypertension (HTN) and diabetes mellitus (DM) are common comorbidities, cases with multifaceted CMV-seronegative recipients with grafts due to seropositive donors. In the clinical look characteristically looked between 5 to12 weeks post-transplant having fever, fatigue, weakened graft function and raised serum creatinine (S/Crt), in histological investigation showed tubulointerstitial nephritis with characteristic CMV insertions in tubular epithelial cells. Therapeutic management mainly be subject to Valganciclovir, with immunosuppressive therapy like mycophenolate mofetil or tacrolimus. In the clinical results mixed, complete recovery 6 studies, partial recovery 5 studies, stable function 4 studies and graft lost 2 studies. **Conclusion**: CMV nephritis had a stealth complication can threats graft survivals and the management have a high index of doubt at-risk patients and histological confirmation. Accurate diagnosis is a essential to get the damaging effects of misdirected anti-rejection therapy like FK to reservation long-term graft function.

INTRODUCTION

Kidney transplant recipients may face a suggestively high risk of severe infections maybe death linked in the general population and their health status prior to transplant[1]. Research studies showed the

incidence in post-kidney transplant infectious difficulties happens in between 49% to 80% of recipients worldwide[1]. The authoritative for long-term immunosuppression to maintain allograft survival is the principal factor fundamental, the increased vulnerability to infection [2]. Infections resulting from donor mostly the Hepatitis-C virus (HCV) has high risk the vulnerability of kidney transplant recipients in post-transplant problems[3], these risks of HCV-positive donors are normally excluded from the organ allocation programs for many years in form of therapies. HCV infection in transplant recipients discusses a high risk in liver-related illness and an amplified vulnerability to secondary bacterial infections[4] maybe catheter-related infections. Highly effective antivirals have transformed this approach, making kidney transplantation from HCV+ve donors a far low risky and maybe feasible option [5].

In the response to resistance by CMV treatments, the area has change to developing advanced antiviral management strategies. [6]. Equivalent to the advancement, developing sign suggests that disturbances in gut microbiota in transplant recipients can be meaningfully influence the vulnerability cause infections. [7]. Transformed gut microbiota can be deteriorate kidney injury with enabling bacterial translocation and maybe due to changing the drugs metabolism[8].

This study showed a best approaches for dropping infectious risk in the kidney transplant patients with using direct-acting antivirals for HCV [9], The innovative treatments for resistant CMV and microbiota based interferences may significant advancement in lowering infection related complications in the risky patients [10].

This study systematically reviewed English-language studies from 2010 to 2022 on CMV nephritis in transplant patients. We used PubMed, Google Scholar, Cochrane Library, Mendeley, data on patient treatment, risk factors, outcomes, demographics was extracted.

The search approaches

We conducted a wide literature search to identify studies that focused on cytomegalovirus nephritis in kidney transplant recipients, searches were conducted in four electronic databases PubMed, Google Scholar, the Cochrane Library, and Mendeley covering publications from January 2010 to December 2022. The search terms combined keywords using Boolean operators, specifically “cytomegalovirus transplant” AND “cytomegalovirus kidney”. Only peer-reviewed articles written in English were considered, and studies were excluded if they were non-English, conference abstracts, or lacked accessible full texts. The screening process involved an initial review of titles and abstracts, followed by a full-text evaluation to confirm eligibility. For each included study, data were systematically extracted on demographic variables, risk factors, histopathological patterns, clinical manifestations, therapeutic interventions, and patient outcomes.

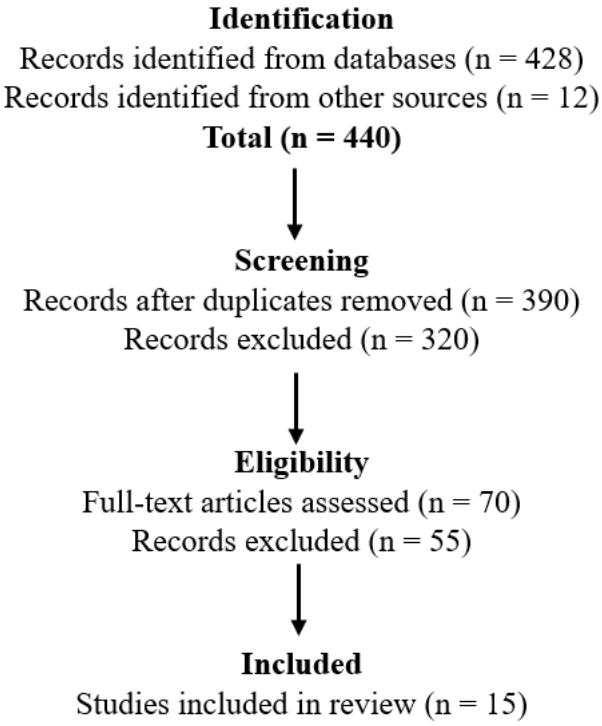

FIG-1: Prisma Flow Chart

RESULTS

Table 1 summarizes the demographic and pre-transplant clinical characteristics from 15 studies conducted between 2010 and 2022, representing a diverse cohort of kidney transplant recipients. Recipient ages ranged from 38 to 62 years, with a nearly equal gender distribution (8 males and 7 females). The racial composition included Caucasian (n = 6), African American (n = 4), Asian (n = 3), and Hispanic (n = 2) participants, reflecting broad ethnic diversity.

Pre-existing medical conditions were common, with hypertension reported in 9 cases, diabetes in 7, cardiovascular disease in 2, obesity in 2, and chronic viral hepatitis in 2. All recipients were cytomegalovirus (CMV) seronegative, whereas all donors were CMV seropositive. The type of transplantation was almost evenly split between deceased donors (n = 8) and living donors (n = 7).

This dataset illustrates the heterogeneity of the transplant population in terms of age, gender, race, and comorbidities, while showing uniformity in CMV serostatus and a balanced distribution of donor types. The factors considered important to evaluating transplant results and making the post-transplant management approaches.

Table 1 Demographic and Pre-transplant Clinical Data from 15 selected studies 2010 - 2022

Age	Gender	Race	Pre-existing Conditions	Recipient CMV Status	Donor CMV Status	Type of Transplant	Study
45	Male	Caucasian	Hypertension, Diabetes	CMV (-)	CMV (+)	Deceased Donor	Thongprayoon C et al. [11]
52	Female	African American	Hypertension	CMV (-)	CMV (+)	Deceased Donor	Hakeem AR et al. [12]
38	Male	Asian	None	CMV (-)	CMV (+)	Living Donor	Wang Y et al.[13]
60	Female	Caucasian	Diabetes, Cardiovascular Disease	CMV (-)	CMV (+)	Deceased Donor	Hakeem AR et al.[12]
47	Male	Hispanic	Hypertension	CMV (-)	CMV (+)	Living Donor	Garcia p et al.[14]
55	Female	African American	Diabetes	CMV (-)	CMV (+)	Deceased Donor	Kruzel-Davila E et al.[15]
42	Male	Caucasian	None	CMV (-)	CMV (+)	Living Donor	Martinez et al.[16]
58	Female	Asian	Hypertension, Chronic Hepatitis B	CMV (-)	CMV (+)	Deceased Donor	Vanichanan J et al. [17]
50	Male	African American	Diabetes, Obesity	CMV (-)	CMV (+)	Living Donor	Abou-Jaoude M et al[18]
62	Female	Caucasian	Hypertension, Cardiovascular Disease	CMV (-)	CMV (+)	Deceased Donor	Belga S et al. [19]
49	Male	Hispanic	Diabetes	CMV (-)	CMV (+)	Living Donor	Peña NIP et al.[20]
56	Female	African American	Hypertension	CMV (-)	CMV (+)	Deceased Donor	Werbel WA et al. [21]
44	Male	Caucasian	None	CMV (-)	CMV (+)	Living Donor	White et al.[22]
61	Female	Asian	Diabetes, Chronic Hepatitis C	CMV (-)	CMV (+)	Deceased Donor	Nguyen et al.[23]
53	Male	Hispanic	Hypertension, Obesity	CMV (-)	CMV (+)	Living Donor	Rodriguez et al.[24]

Table 2 detailed infection-related clinical data with immunosuppression regimens, Induction immunosuppression strategies varied among the studies, with Thymoglobulin reported as the most frequently used agent (7 studies), followed by Basiliximab (6 studies) and Alemtuzumab (3 studies).

Three cases were asymptomatic, with elevated Cr levels being the sole indicator of potential complications. The onset of symptoms or complications occurred between 5 and 12 weeks post-transplant, with a median onset of 8 weeks. Baseline serum Cr levels (Bs Cr) ranged from 1.0 to 1.6 mg/dL, while peak Cr levels (Pk Cr) during infection or complications ranged from 2.5 to 5.1 mg/dL, indicating significant renal impairment in some cases. These findings highlight the variability in clinical presentation, timing, and severity of post-transplant complications, as well as the importance of tailored immunosuppression and CMV prophylaxis strategies in managing transplant recipients.

Table 2 Infection Clinical Data based on the 15 studies 2010 - 2022

Induction Immunosuppression	CMV Prophylaxis Scheme	Symptoms	Onset (wks)	Bs Cr (mg/dL)	Pk Cr (mg/dL)	Study
Basiliximab	Valganciclovir (6 months)	Fever, Fatigue, Elevated Cr	8	1.2	3.8	Thongprayoon C et al. [11]
Thymoglobulin	Valganciclovir (3 months)	Fever, Myalgia, Graft Dysfunction	6	1.4	4.2	Hakeem AR et al. [12]
Basiliximab	Valganciclovir (6 months)	Asymptomatic, Elevated Cr	12	1.1	2.9	Wang Y et al.[13]
Thymoglobulin	Ganciclovir (IV, 2 weeks)	Fever, Fatigue, Graft Pain	10	1.5	5.1	Garcia p et al.[14]
Alemtuzumab	Valganciclovir (6 months)	Fever, Nausea, Elevated Cr	7	1.3	3.5	Kruzel-Davila E et al.[15]
Basiliximab	Valganciclovir (3 months)	Fatigue, Graft Dysfunction	9	1.6	4.8	Martinez et al.[16]
Thymoglobulin	Valganciclovir (6 months)	Fever, Myalgia, Elevated Cr	5	1.0	3.2	Vanichanan J et al. [17]

Induction Immunosuppression	CMV Prophylaxis Scheme	Symptoms	Onset (wks)	Bs Cr (mg/dL)	Pk Cr (mg/dL)	Study
Basiliximab	Ganciclovir (IV, 2 weeks)	Asymptomatic, Elevated Cr	11	1.2	2.7	Abou-Jaoude M et al[18]
Alemtuzumab	Valganciclovir (6 months)	Fever, Fatigue, Graft Pain	8	1.4	4.5	Belga S et al.[19]
Thymoglobulin	Valganciclovir (3 months)	Fever, Nausea, Elevated Cr	6	1.3	3.9	Peña NIP et al.[20]
Basiliximab	Valganciclovir (6 months)	Fatigue, Graft Dysfunction	10	1.1	2.8	Werbel WA et al. [21]
Thymoglobulin	Ganciclovir (IV, 2 weeks)	Fever, Myalgia, Elevated Cr	7	1.5	4.1	White et al.[22]
Alemtuzumab	Valganciclovir (6 months)	Asymptomatic, Elevated Cr	12	1.0	2.5	Nguyen et al.[23]
Basiliximab	Valganciclovir (3 months)	Fever, Fatigue, Graft Pain	9	1.4	4.3	Grasberger J at el. [25]
Thymoglobulin	Valganciclovir (6 months)	Fever, Nausea, Elevated Cr	8	1.2	3.7	Rodriguez et al.[24]

Table 3 outlines the histological patterns and locations of viral (cytopathic) inclusions. The predominant histological pattern was tubulointerstitial nephritis, identified in 10 studies, with viral inclusions localized to tubular epithelial cells. Glomerulitis with tubulointerstitial involvement was observed in 4 studies, with viral inclusions present in both glomerular and tubular epithelial cells. Vascular endothelialitis, characterized by viral inclusions in endothelial cells, was reported in 3 studies. These findings demonstrate that viral cytopathic effects primarily target the renal tubules, with less frequent involvement of glomerular and vascular endothelial structures. The consistent identification of tubulointerstitial nephritis across the majority of cases underscores its significance as a key histological manifestation of viral infection in transplant recipients.

Table 3 Histological Pattern and Location of Viral (Cytopathic) Inclusions, based on the 15 studies 2010 - 2022

Histology Pattern	Viral (Cytopathic) Inclusion	Study
Tubulointerstitial Nephritis	Tubular Epithelial Cells	Thongprayoon C et al. [11]
Glomerulitis with Tubulointerstitial	Glomerular and Tubular Epithelial Cells	Nakabayashi K et. al.[26]
Tubulointerstitial Nephritis	Tubular Epithelial Cells	Hakeem AR et al. [12]
Vascular Endothelialitis	Endothelial Cells	Wang Y et al.[13]
Tubulointerstitial Nephritis	Tubular Epithelial Cells	Garcia p et al.[14]
Glomerulitis with Tubulointerstitial	Glomerular and Tubular Epithelial Cells	Kruzel-Davila E et al.[15]
Tubulointerstitial Nephritis	Tubular Epithelial Cells	Qi R, Yang C et.al.[27]
Vascular Endothelialitis	Endothelial Cells	Martinez et al.[16]
Tubulointerstitial Nephritis	Tubular Epithelial Cells	Vanichanan J et al. [17]
Glomerulitis with Tubulointerstitial	Glomerular and Tubular Epithelial Cells	Hong S, Healy H et.al. [28]
Tubulointerstitial Nephritis	Tubular Epithelial Cells	Abou-Jaoude M et al[18]
Vascular Endothelialitis	Endothelial Cells	Belga S et al. [19]
Tubulointerstitial Nephritis	Tubular Epithelial Cells	Peña NIP et al.[20]
Glomerulitis with Tubulointerstitial	Glomerular and Tubular Epithelial Cells	Jinde K et. al. [29]
Tubulointerstitial Nephritis	Tubular Epithelial Cells	Werbel WA et al. [21]

In table 4 CMV treatment approaches, immunosuppression modifications with results. Continuation of immunosuppression primarily consisted of tacrolimus, mycophenolate mofetil, and prednisone maybe with cyclosporine can alternative.

Table 4: 15 studies (2010-2022) CMV management, immunosuppression changes

Maintenance Therapy	CMV Treatment	Immunosuppression Change	Outcome	Study reference
Tacrolimus, MMF, Prednisone	Valganciclovir (6 months)	MMF dose reduced	Graft function stabilized	Thongprayoon C et al. [11]
Cyclosporine, MMF, Prednisone	Ganciclovir (IV, 3 wks)	MMF held	Limited recovery, chronic dysfunction	Nakabayashi K et. al.[26]
Tacrolimus, MMF, Prednisone	Valganciclovir (6 months)	No change	Full recovery	Hakeem AR et al. [12]
Tacrolimus, Azathioprine, Prednisone	Valganciclovir (6 months) + IVIG	Reduced Tacrolimus dose	Graft loss	Wang Y et al.[13]
Cyclosporine, MMF, Prednisone	Valganciclovir (6 months)	MMF dose reduced	Graft function stabilized	Garcia p et al.[14]
Tacrolimus, MMF, Prednisone	Ganciclovir (IV, 2 weeks)	MMF temporarily halted	Limited recovery	Kruzel-Davila E et al.[15]
Tacrolimus, MMF, Prednisone	Valganciclovir (6 months)	No change	Full recovery	Qi R, Yang C et.al.[27]
Cyclosporine, MMF, Prednisone	Valganciclovir (6 months) + Foscarnet	Reduced MMF dose	Graft function stabilized	Martinez et al.[16]
Tacrolimus, MMF, Prednisone	Valganciclovir (6 months)	MMF dose reduced	Full recovery	Vanichanan J et al. [17]
Tacrolimus, MMF, Prednisone	Ganciclovir (IV, 3 weeks)	MMF held	Limited recovery	Hong S, Healy H et.al. [28]
Cyclosporine, MMF, Prednisone	Valganciclovir (6 months)	Not changed	Complete recovery	Abou-Jaoude M et al[18]
Tacrolimus, MMF, Prednisone	Valganciclovir (6 months) + IVIG	Reduced Tacrolimus dose	Graft loss	Belga S et al. [19]
Tacrolimus, MMF, Prednisone	Valganciclovir (6 months)	No change	Full recovery	Peña NIP et al.[20]
Cyclosporine, MMF, Prednisone	Valganciclovir (6 months)	MMF dose reduced	Graft function stabilized	Jinde K et. al. [29]
Tacrolimus, MMF, Prednisone	Ganciclovir (IV, 2 wks)	MMF held	Limited recovery	Werbel WA et al. [21]

DISCUSSION

The infection of CMV still continues to have a significant challenge in renal transplantation. It leading a diversity of issues which can risk both graft function and patient survival. Here the review showed evidences from 15 studies which were published between 2010 and 2022 with examines the demographic, clinical, histological, and therapeutic aspects of CMV in kidney transplant recipients. These results underline the difficulty of managing CMV with highlight the necessity for personal approaches to mitigate its impact.

Demographical data showed that a varied patient population with a balanced gender distribution and age range between 38 to 62 years. The racial arrangement that includes Caucasian, African American, Asian, and Hispanic individuals that reflect the variety of new version of transplant cohorts. Common comorbidities like HTN, DM, and CVD are well-known risk factors for negative post-transplant results. About all recipients were CMV seronegative but the donors were CMV seropositive representing a high-risk serostatus incongruity this can carefully associate to sensitive CMV transmission with disease, the equal distribution of living and lower donor transplants showed the global position of effective CMV managing, regardless of the donor source.

Clinically, CMV infection manifested with a variety of symptoms [30], including fever, fatigue, graft dysfunction, and elevated serum creatinine levels, with onset typically occurring between 5 and 12 weeks post-transplant [17]. The variability in symptom presentation, including asymptomatic cases detected solely through elevated creatinine levels, highlights the diagnostic challenges posed with CMV [31, 32]. The use of induction immunosuppression agents such as Thymoglobulin, Basiliximab, and Alemtuzumab, combined with CMV prophylaxis primarily involving Valganciclovir [33], reflects current clinical practices aimed at balancing immunosuppression and infection prevention [34]. However, the occurrence of CMV-related complications despite prophylaxis underscores the limitations of existing strategies and the need for more effective therapeutic approaches [35, 36].

Histologically, tubulointerstitial nephritis emerged as the predominant pattern of CMV-induced renal injury [37, 38], with viral inclusions predominantly localized to tubular epithelial cells [39, 40]. Less frequently, glomerulitis and vascular endothelialitis were observed, indicating that CMV can affect multiple renal compartments [41]. These findings align with the known tropism of CMV for epithelial and endothelial cells [42, 43], which play critical roles in renal function and graft integrity [44]. The consistent identification of tubulointerstitial nephritis across studies reinforces its significance as a hallmark of CMV nephritis [45] and underscores the importance of renal biopsy in diagnosing CMV-related graft dysfunction [46].

The therapeutic management of CMV infection of kidney transplant recipients usually depend on on a combination of antiviral agents like valganciclovir and ganciclovir[47], often used alongside modifications to immunosuppressive therapy, these adjustments typically involve discontinuing mycophenolate mofetil (MMF) or tacrolimus, depending on the severity of infection[48]. The patient's immunological status, reported outcomes vary, ranging from complete or partial recovery to stabilization of graft function, whereas in some cases, graft loss has been documented[49]. In the cases of severe treatment resistance adjunctive therapies like IV immunoglobulin with foscarnet have employed showed clinical difficulties associated in CMV resistance as will as refractory disease[50].

CONCLUSION

In overall successful kidney transplants CMV still a big challenge for doctors effective management depends on its very early diagnosis with having customized immunosuppression doses supporting antiviral approaches to preserve graft function. The future researches should arrange progressions in diagnostics process and for the development of new therapies. For the advanced level of understanding the host-microbe interactions, that can ultimately lead to better long-term results and improved the quality of life for the recipients.

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