



Immunology in Kidney Transplantation

Juan Carlos H Hernandez Rivera*

Department of Nephrology, Mexican Institute of Social Security, Mexico

*Corresponding author: Juan Carlos H Hernandez Rivera, Department of Nephrology, Mexican Institute of Social Security, Mexico, Tel: +52 800 623 2323; E-mail: juancarloshhernandezrivera@hotmail.com

Received date: September 25, 2017; Accepted date: September 27, 2017; Published date: October 5, 2017

Copyright: © 2017 Hernández-Rivera JCH. This is an open-access article distributed under the terms of the Creative Commons Attribution License; which permits unrestricted use; distribution; and reproduction in any medium; provided the original author and source are credited.

Citation: Hernandez-Rivera JCH (2017) Immunology in Kidney Transplantation. Immunol Curr Res 1: e102.

Editorial

Chronic kidney disease (CKD) is a public health problem, due to its high incidence and prevalence, especially associated with chronic-degenerative diseases such as diabetes mellitus (DM) and systemic arterial hypertension (SAH). However, these causes, although they represent the majority of the etiology, are not the only ones. There are some others, such as glomerular primary, secondary glomerular diseases, vascular or urinary system malformations, polycystic disease, and infectious diseases, many others. Because of the problem that CKD represents, substitution therapies to lengthen the survival of a person have become more important and transcendent, with Renal Transplantation (RT) the one that offers the greatest survival in patients [1,2].

The functionality of the renal graft, throughout the experience, depends on many already established factors and not many others. Immunology in renal transplantation has been a constant advance and continues to contribute multiple findings favoring a greater survival of the renal grafts, from the ABO system, where efforts for transplantation with group incompatibility have been seen as a reality based on the iso-agglutinin and subgroup studies [3,4] to the study of the human leukocyte antigen (HLA) where the incorporation in the panel of alleles included in the basic study of the receptor-donor binomial has increased, in the last decade considering the locus Cw and the PD as some cause of rejection that previously did not had been considered.

It has recently been deepened in search of greater acceptance of the immune system so that more progress is made in this field, a few years ago the study of donor-directed antibodies, called Specific Donor Antibodies (ADE), complement fixation (with the activation of the classical pathway by C1q) as well as knowing which immunoglobulin G may be more relevant, all to better understand the biochemical mechanisms involved. The methodology for detecting antibodies has also evolved in recent years and Luminex methods and flow cytometry are able to detect antibodies with extremely low titers; another of the conditions that influence the defense of the host is the C-reactive protein, largely dependent on the ability to activate C1q mainly given by asparagine and histidine, which allow the correct assembly to initiate the complement cascade and forms part of the immune system that contributes to the defense of the first-line host, inflammation events, in addition to T-cell immunity, CD4 + and CD8 + T cell response models [5-7].

The behavior in antibody-mediated rejection emphasizes the dependence of anti-HLA ADEs, as well as complement activation,

being associated with graft dysfunction and transplant loss in a significant way. In addition, the effort to establish the predictive power of the complement-fixing ADEs, specifically C1q and C3d, correlating with the deposits in the C4d renal graft biopsy and culminating with the onset of the membrane attack complex in the renal graft [8,9].

This does not conclude here, being indispensable to search from the knowledge of the signaling pathways of the transplant as mentioned more than a decade ago by Halloran, seeking the selective blocking of the three described pathways and their new potentials of these; we are in a time where efforts in the molecular field must prevail for the greater success of kidney transplants and thereby guarantee our patients a better survival of the graft and with it a better quality of life for them and their families.

References

1. Avila-Saldivar MN (2013) Enfermedad renal cronica: prevencion y detección temprana en el primer nivel de atencion. *Med Int Mex* 29: 148-153.
2. Torrez-Salazar J, Torrez-Salazar J, Espinoza-Dávila E, Gutierrez-Méndez J (2011) Sobrevida del injerto renal en un centro hospitalario. *Gac Med Bol* 34: 87-90.
3. Musso C, Giordani M, Imperiali N (2016) Aging Kidney Transplantation. *Rev Inves Clin* 68: 68-74.
4. Ghoneim M, Bakr MA, Refaie AF, Akl AI, Shokeir AA, et al. (2013) Factors affecting graft survival among patients receiving kidneys from live donors: a single-center experience. *Biomed Res Int* 912413.
5. Timckam K, Heeger P (2013) Transplantation: Complementing donor-specific antibody testing. *Nat Rev Nephrol* 9: 713-717.
6. Loupy A, Lefaucheur C, Vernerey D, Prugger C, Huyen JPD, et al. (2013) Complement-binding Anti-HLA antibodies and Kidney-Allograft survival. *N Engl J Med* 369: 1215-1226.
7. Agrawal A, Shrive AK, Greenhough TJ, Volanakis JE (2001) Topology and structure of the C1q-binding site on C-reactive protein. *J Immunol* 166: 3998-4004.
8. Sicard A, Ducreux S, Rabeyrin M, Couzi L, Mc Gregor B, et al. (2015) Detection of C3d-Binding Donor-Specific Anti-HLA Antibodies at Diagnosis of Humoral Rejection Predicts Renal Graft Loss. *J Am Soc Nephrol* 26: 457-467.
9. Frank R, Lal P, Kearns J, Molina MR, Wald JW, et al. (2016) Correlation of Circulating Complement-Fixing Donor-Specific Antibodies Identified by the C1q Assay and Presence of C4d in Endomyocardial Biopsy Specimens. *Am J Clin Pathol* 145: 62-68.
10. Penn H, Howie AJ, Kingdon EJ, Bunn CC, Stratton RJ, et al. (2007) Scleroderma renal crisis: patient characteristics and long-term outcomes. *QJM* 100: 485-494.