J Clin Cell Immunol 2015, 6:5
http://dx.doi.org/10.4172/2155-9899.C1.025

OMICS International
conferenceseries.com

4th International Conference and Exhibition on
Immunology
September 28-30, 2015 Crowne Plaza Houston River Oaks, Houston, TX, USA

Evaluation of chronic allograft dysfunction in renal transplant recipients- Single centre analysis

Sailaja Kesiraju
Bhagwan Mahavir Medical Research Centre, India

Despite improvement in the short term graft survival due to progress in controlling acute rejections, improvement in the immunosuppression, chronic rejection remains a considerable hindrance, preventing the goal of long term graft survival. Chronic allograft dysfunction (CAD) or chronic allograft nephropathy (CAN) is the major cause of long-term graft loss in kidney transplant recipients.CAD is defined as functional and morphologic deterioration of a renal allograft. CAD is characterized clinically by progressive deterioration in renal function combined with proteinuria, hyper tension and renal insufficiency with nonspecific pathology in renal transplant recipients. Chronic allograft dysfunction is the most prevalent cause of late renal allograft loss. A complex network of cellular mechanisms in both graft and peripheral immune compartments complicates the non-invasive diagnosis of CAN, thereby requiring biopsy histology. This is compounded by non-immunological factors contributing to graft injury. Immunological factors include: Chronic active rejection, chronic rejection of both cellular and humoral, while non immunological include CNI toxicity, recurrence of glomerular disease, infections etc. Chronic rejection is not reversible and has been recognized as the most important cause of the graft dysfunction. Chronic rejection is a complex pathophysiological response involving many components of the immune system as well as non-immune factors. T cells, macrophages, chemokines, pro-inflammatory cytokines and alloantibodies have all been implicated in both the initiation and progression of the rejection process. We aimed to assess the prevalence and correlation factors of graft dysfunction, and its effects on long-term graft survival. Renal transplant recipients transplanted between March 2000 and April 2014 were included. Clinical parameters, etiology of ESRD, number of dialysis, surgical complications, history of acute rejections, creeping serum creatinine, immuno-suppression regimen, glomerular filtration rate, and patient and graft survivals were analyzed. CAD and death with functioning graft were the major causes of the graft loss in this cohort. Recurrence of the glomerular disease was the third major cause of graft loss. Acute tubular necrosis was the most common cause for renal graft dysfunction. High dose of CNIs in the early stage might affect the recovery of the early renal function. The early renal dysfunction, donor age and AR episodes would affect the renal graft long-term survival.

kesirajusailaja@gmail.com

Response of cancer cell lines to small molecules (cytotoxicant) – Resulting in apoptosis and autophagy

Shailasree S
University of Mysore, India

Systematic screening of chemical libraries for small molecules revealed limited studies involving promising small molecules (lacking details on studies inferring drug-target interactions) with a capacity to kill cancer cells in-vitro. In the present study, myricetin was evaluated for its capacity to induce cytotoxic effect to neuroblastoma N2a cell and have listed the multi-target paradigm leading to its growth inhibition, apoptosis/autophagy. Toxic pathway, especially the upstream network of responses happening in toxicant-treated cancer cells prior to their programmed cell death is reported to provide an unbiased approach in unraveling changes deciding on the final fate of the cell. Studies preceding cell death by probe sets strongly pointed to changes in cluster related to genes with a role in chromosomal stability, e.g., heterogeneous nuclear ribonucleoprotein (*HNRNPM*), that was down regulated. Those involved in adaptive carbon metabolism e.g., argininosuccinate synthase (ASS1) were upregulated identified as intermediate response upon exposure to toxicant. Consumption of phosphocreatine and a parallel accumulation of creatine indicated exhaustion of cellular energy buffer. The prominent role of GSH to counter increasing cell stress as early adaptation before breakdown of cellular homeostatis was observed. Direct data substantiating cell death by apoptosis with p38 MAP kinase mediated p53 activated upregulation of caspase 3 is reported and will be discussed. Our data report on autophagy, representing an additional mechanism inducing cell death detected by accumulation of LC3-II protein and acridine-orange stained autophagosomes is included.

shailasree_s@yahoo.co.uk