Mohammad Husain, Health Care: Current Reviews 2015, 3:3
http://dx.doi.org/10.4172/2375-4273.C1.011

OMICS International
conferenceseries.com

3rd Indo-Global Summit & Expo on
Healthcare
October 05-07, 2015 New Delhi, India

Role of vitamin D receptor (VDR) in HIV-1 induced kidney injury

Mohammad Husain[1,2]
[1]Jamia Millia Islamia, India
[2]Feinstein Institute for Medical Research, USA

Observational studies have demonstrated significant low levels of vitamin D in HIV-infected patients. Antiretroviral drugs such as Protease inhibitors (PI) and Non Nucleoside Reverse Transcriptase Inhibitors (NNRTI) exert metabolic interference with vitamin D. Biological action of vitamin D depends on the expression and nuclear activation of vitamin D receptor (VDR). In addition, VDR also regulates a plethora of genes. To understand the role of VDR in kidney injury, we hypothesized that HIV-induced reactive oxygen species (ROS) may cause kidney cell injury through downregulation of VDR. To explore the role of HIV-induced ROS on downregulation of VDR, an in vitro analysis was performed using proximal tubular cells. It was observed that HIV downregulated VDR in addition to inflicting DNA damage. These effects, however, were reversed when EB-1089 (a VDR agonist, VD) was used. To confirm the role of HIV-induced ROS in downregulation of VDR, H2O2 (an O- donor) was used on normal tubular cells that directly downregulated tubular cell VDR. The use of catalase, a free radical scavenger restored VDR expression in both HIV-induced and H2O2 treated tubular cells. HIV also stimulated the tubular cell rennin-angiotensin system (RAS) through downregulation of VDR. Since losartan (an ANGII blocker) partially inhibited HIV-induced ROS generation in tubular cells, it appears that HIV-induced ROS production was contributed partly by RAS activation. The administration of VD in tubular cells, however, not only reduced HIV-induced RAS activation but also attenuated tubular cell ROS generation and thus restoring VDR expression. The findings indicate that HIV-induced tubular cell downregulation of VDR contributed to the RAS activation and associated DNA damage. Addition of VDand RAS blockade provided protection against these effects of HIV-1.

Biography

Mohammad Husain is an Associate Professor, Department of Biotechnology, JMI, New Delhi. He is a Visiting Professor at Feinstein Institute for Medical Research, New York and Senior Research Scientist, Feinstein Institute for Medical Research, North Shore-LIJ Health System, Manhasset, NY.

mhusain2@jmi.ac.in

Notes: