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Skewing of SAG mediated therapy for a predominant Th1 during Visceral Leishmaniasis on triggering CD2 epitope to circumvent liver infection

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Introduction : Visceral leishmaniasis is a macrophage associated disorder which is linked with a profound decrease in the immunotherapeutic potential of the infected subjects leading to a marked reduction in the CD4 linked Th1 protective immune response. It greatly affects the liver leading to abnormal levels of SGPT and SGOT. Also the patients suffering from VL have been reported to be coinfected with Hepatitis C during some circumstances. Simultaneously the patients in Bihar are showing unresponsiveness towards SAG which is still a first line of drug in many countries around the world against Visceral Leishmaniasis. .

Methods : The T cells were isolated from patients and were subjected to FACS analysis and cytokine analysis.

Results : We have reported down regulation of CD2 co receptor on the surface of CD4 cells in patients suffering from Visceral Leishmaniasis. Stimulation of CD2 epitope with antiCD2 antibody has led to a remarkable increase in the Protein kinase C alpha mediated phosphorylation on CD2 co receptor on CD4 T cells, induction of IFN-γ led Th1 dominated immune response, a substantial increase in the lymphoblast population and this response remained Th1 dominated even in the presence of Th2 predominant conditions signified with rIL4.

Conclusions : We have shown that biological immunomodulators such as interferon (IFN)-γ can provide a missing signal and enhance the activity of antimonials in the treatment of VL and CL

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