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Challenges due to variation in pathogenic affinity to host cell receptors in management and control of HIV/AIDS

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Human immunodeficiency virus (HIV) is highly variable due to its poor proof reading activity which results in to presence of distinct and multiple variants in different cells and secretions of the same individual. These variants may have variable affinity to different immune cells and therefore may influence the disease progression. Additionally HIV has been demonstrated to bind different host cell receptors including human Mannose Receptor (hMR), CD-SIGN, Galactosyl Ceramide Syndecan-Syndecan-3 Heparan Sulfate Proteoglycan receptors. Sexually HIV has been transmitted by binding to hMR on human sperm, vaginal epithelial which are devoid of conventional CD4 receptor and are responsible for sexual transmission of HIV. HIV binding to hMR induces Matrix Metallo-proteinase 9 (MMP9) which weakens the cell surface and therefore increase the risk for sexual transmission of HIV. Further the localization of hMR was found to be in lower number of vaginal epithelial cells of HIV negative female partner of serodiscordant couples as compared to normal females suggesting the association of hMR in sexual transmission of HIV. Genotypic characterization of C2-V3 region of HIV1 C *env* gene in PBMCs, sperm, vaginal epithelial cells and cervical cells showed presence of distinct variants in the same individual with variable infectivity. These variants showed different numbers of N-linked glycosylation (NLG) sites suggesting variation in co-receptor affinity in different cells for the same individual which may be associated with progression to disease and also with risk of sexual transmission of HIV. The study suggests association of hMR in sexual transmission of HIV. CD4+CD25 (IL-2 receptor)+CD127 (IL-7 receptor)-FOXP3+Treg cells were investigated from Peripheral Blood Mononuclear Cells (PBMC) healthy, HIV-1 and HIV-2 infected individuals on anti-retroviral therapy (ART) demonstrated increase in CD4+CD25+CD127-Treg cells few of the HIV-1 infected individuals as compared to healthy controls and HIV-2 infected individuals. Presence of distinct HIV variants in PBMCs and urogenital cells which may influence the viral affinity to host and immune cells therefore may affect the transmission, infectivity and pathogenicity. These variable host and pathogenic responses remain the challenges in designing the strategies for management of pathogenesis and sexual transmission of HIV.

Some biochemical markers that can predict pre-eclampsia

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TNF-α directly damages the vascular endothelial cells, reduces regional blood flow, causes occlusion of vessels and increases endothelial permeability. Endothelial cell injury after TNF-α mediated activation of immune system may result in secretion of vasoactive substances and increase in vascular permeability and intravascular coagulation. TNF-α may be involved in the pathogenesis of preeclampsia and may identify the patients who are at high risk of PE and can be a potential marker of the severity of the preeclampsia syndrome. Women with preeclampsia had deranged lipid profile due to abnormal lipid metabolism; this alteration of lipid metabolism may play a key role in the development of symptoms of Pre-eclampsia. Furthermore, changes to lipid metabolism may contribute towards the endothelial lesions observed in pre-eclampsia.