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HIV and viral hepatitis among high-risk groups in the Middle East and North Africa: Understanding the knowledge gap

Nada M Melhem

American University of Beirut, Lebanon

It is well established that human immunodeficiency virus (HIV)-positive individuals co-infected with hepatitis B virus (HBV) or hepatitis C virus (HCV) suffer from liver pathology associated with morbidity and mortality. Moreover, HIV-infected individuals do not respond well to treatment for HBV or HCV and hence are at increased risk of hepatic toxicity. Consequently, co-infection of HIV-positive individuals with HBV and/or HCV is a global health problem of significant magnitude. Little is known about the co-infection status of HIV, HBV and HCV in the Middle East and North Africa (MENA) region. HIV, HBV and HCV are blood-borne viruses with similar modes of transmission. The categories of people at high risk of acquiring HIV-1, HBV and HCV commonly include: Men who have sex with men (MSMs), female sex workers (FSW), injecting drug users (IDUs). The aim of this review is to identify gaps in the existing knowledge on single, dual and triple infections of HIV, HBV and HCV in the region among MSMs, FSWs, IDUs and prisoners. This review highlights the paucity and the variability of existing data on high-risk groups and the status HIV, HBV, HCV infections and co-infection in the MENA region. Without addressing the risks of expanding epidemics among high-risk groups, an AIDS free society will remain an illusion. It is obvious that resources need to be allocated to inform strategic planning and policy of the silently creeping waves of HIV and viral hepatitis epidemics among these groups.

melhemn@aub.edu.lb

Humanized mice to study HIV-1 clade specific differences in course of infection and neuropathology

Santhi Gorantla

University of Nebraska Medical Center, USA

Background: Understanding the role-played by HIV-1 clades in viral transmission and disease pathobiology remains far from complete. NOD/scid-IL-2R γ_c^{null} (NSG) mice reconstituted with functional human immune system allows for productive long-term viral infection and associated immune and central nervous system pathologies. Thus, humanized NSG mice were used to assess clade-specific differences in viral virulence, cytopathicity and neurotoxicity.

Methods: NSG mice (reconstituted at birth with human CD34+ stem cells) were infected either with HIV-1 clade B or C strains. Blood viral load (VL), CD4 and CD8 T cell numbers, and markers of immune activation were assessed. Brains were collected for quantitative immunohistology and neuropathologic tests.

Results: Evidence of high viral replication and slower CD4+ T cell depletion rate was observed in clade C infections. T cell activation (CD4+CD38+) was notably higher in clade C, while T cell exhaustion (CD4+PD-1+) was lower in clade C correlating to higher number CD4+Ki67+ cells in spleen. Increased immune cell infiltration into the brains of mice with infection was observed with higher rates and extent of meningitis in clade C infected mice (C60 vs. B40%). Incidence of neuropathology in different regions of brain was comparable amongst both clades.

Conclusion: Higher immune activation and T cell replication status in clade C infection might be contributing to both slower rate of CD4+ T cell depletion and brain immune cell infiltration. Humanized mice can be used to provide important insights and explanations needed to understand the disparities seen in viral transmission rate and HAND incidence in different HIV-1 clades.

sgorantla@unmc.edu