

Oral presentation

O211 HIV and the brain: neuro-inflammation long-term, CNS control of HIV replication, brain disease in suppressed patients and differential penetration of drugs

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The central nervous system is involved early in the course of HIV and without treatment frequently is seriously damaged resulting in a serious HIV-associated dementia. This is believed to be due to direct toxicity of viral proteins and indirect damage from inflammatory reaction in the brain over long periods of time. The advent of highly active antiretroviral therapy has almost eliminated severe dementia in the treated HIV population. However, careful examination of patients in our clinics continues to show a majority of chronically infected, treated patients suffer measurable neurocognitive impairment. While this condition is mild in most cases, it may impact long-term quality of life, and might suggest early manifestation of accelerated cognitive decline with aging. Preliminary evidence links low level viral replication determined in the CSF of treated patients with inferior neurocognitive outcomes. There is also evidence of residual neuro-inflammatory activity in the CNS of chronically treated patients, a finding consistent with the hypothesis that ongoing infection may still drive pathology in the brain through smoldering inflammatory responses. One available means of improving outcome could be to target HIV therapies for the brain. When HIV therapies are categorized according to their effectiveness in the brain or CSF, recent evidence supports better cognitive outcomes with more highly effective drug regimens. Ongoing research is critical to determine if outcomes of HIV therapy can be improved by selection of drugs with better CNS penetrating properties.