Character of CSF inflammation through the course of HIV infection

ABSTRACT

One of the salient features of HIV infection is immune activation and establishment of a chronic inflammatory state. Systemically, this can be detected in activation profiles of T–lymphocytes and elevated levels of inflammatory biomarkers in blood, and is implicated in the pathogenesis of some of the 'non–AIDS' morbidity of infection, including cardiovascular disease and some neoplasms. Chronic inflammation is likewise detected in the CNS with elevated levels of inflammatory biomarkers and has been speculated to play a role in HIV–related brain injury. To better define the evolving character of central nervous system (CNS) inflammation accompanying HIV infection and compare it to systemic inflammation, we assessed a panel of 10 soluble inflammatory biomarkers in cerebrospinal fluid (CSF) and blood in grouped subjects representing the spectrum of systemic HIV progression, development of CNS injury and viral suppression. We found that HIV infection was associated with a broad CSF inflammatory response through its full course. Its character diverged from that of systemic inflammation, and changed with systemic disease progression and development of neurological injury. Our findings suggest separate evolution of at least two CNS inflammatory components in those without overt HIV–associated dementia (HAD), one related to lymphocytic inflammation that also associated with CSF pleocytosis and HIV RNA levels and another related to macrophage responses that associated with brain injury. Both of these components were then present in HAD in which CSF inflammation was most prominent and also accompanied by blood–brain barrier disruption, setting this clinical presentation apart as an advanced and marked CNS inflammatory process. Suppression of HIV replication by ART or endogenously in elite controllers was associated with reduced CSF inflammation, though not fully to levels found in HIV uninfected controls. Overall, HIV infection is accompanied by a complex evolving pattern of CNS inflammation that likely relates to interactions of progressive systemic immune and inflammatory reactions, CNS virus populations and CNS injury.

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Palabras clave: Líquido cefaloraquídeo; VIH

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Imagen de RM y espectroscopia en 7 Tesla

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RESUMEN

Los estudios clínicos de IRM se realizan generalmente a intensidades de campo de 1.5 o 3 T. El aumento de la fuerza del campo magnético aumenta el tamaño de la señal de IRM, permitiendo registros en una resolución espacial más alta, o en un menor tiempo de exploración con la misma calidad de imágenes. Para la MR espectroscopia (MRS), hay una ventaja adicional que incrementa la resolución espectral, permitiendo la detección de más compuestos, y una cuantificación más precisa. Sin embargo, muchos retos técnicos hay que superar para obtener las ventajas esperadas de los campos magnéticos más altos. Esta presentación revisará los estudios de IRM y MRS del cerebro humano en 7 T. Se discutirán algunos problemas técnicos, así como ejemplo de las aplicaciones en las enfermedades neurodegenerativas (la enfermedad de Huntington) y la esquizofrenia.

MR imaging and spectroscopy at 7 Tesla

ABSTRACT

Clinical MRI studies are generally performed at field strengths of either 1.5 or 3T. Increasing the magnetic field strength increases the size of the MR signal, allowing images to be recorded at higher spatial resolution, or in a shorter scan time with the same quality. For MR spectroscopy (MRS), there is an additional advantage that the spectral resolution increases, allowing more compounds to be detected, and more accurately quantified. However, many technical challenges have to be overcome in order to fully realize the expected advantages of higher magnetic fields. This presentation will review MRI and MRS for studies of the human brain at 7T. Some technical issues will be discussed, as well as example applications in neurodegenerative disease (Huntington's disease) and schizophrenia.

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Palabras clave: Espectroscopia; Resonancia Magnética

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Neuroimagen de las epilepsias genéticas generalizadas (GGE): Nuevos conocimientos sobre los mecanismos subyacentes

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RESUMEN

Mientras que nuestro conocimiento de las causas y mecanismos que subyacen a las epilepsias focales ha evolucionado con el desarrollo de la cirugía de la epilepsia, todavía nuestra comprensión de los mecanismos está rezagada. Los estudios de neuroimagen funcional y estructural de epilepsias generalizada genética humana (GGE) de las últimas dos décadas ha proporcionado importantes conocimientos sobre la alteración del volumen de la sustancia gris, integridad microestructural y conectividad funcional asociado con GGE. Sin embargo, todavía hay una necesidad de correlación de la neuroimagen con electrofisiología y patología para corroborar los mecanismos propuestos, que pueden ser mejor servidos por modelos animales, tales como los modelos de roedor y babuino de GGE.

Neuroimaging of genetic generalized epilepsies (GGE): New insights into underlying mechanisms

ABSTRACT

While our knowledge of the causes and mechanisms underlying focal epilepsies has evolved with the development of epilepsy surgery, our understanding of the mechanisms is still lagging. Functional and structural neuroimaging studies of human Genetic Generalized Epilepsies (GGE) from the past two decades has provided important insights into alteration of gray matter volume, microstructural integrity and functional connectivity associated with GGE. However, there is still a need for correlation of neuroimaging with pathology and electrophysiology to corroborate proposed mechanisms, which may be best served by animal models, such as the rodent and baboon models of GGE.