



Abstracts

Articles appearing in the September 2017 issue

Varicella zoster virus–infected cerebrovascular cells produce a proinflammatory environment

Objective To test whether varicella-zoster virus (VZV) infection of human brain vascular cells and of lung fibroblasts directly increases proinflammatory cytokine levels, consistent with VZV as a causative agent in intracerebral VZV vasculopathy and giant-cell arteritis (GCA).

Methods Conditioned supernatant from mock- and VZV-infected human brain vascular adventitial fibroblasts (HBVAFs), human perineurial cells (HPNCs), human brain vascular smooth muscle cells (HBVSMCs), and human fetal lung fibroblasts (HFLs) were collected at 72 hours postinfection and analyzed for levels of 30 proinflammatory cytokines using the Meso Scale Discovery Multiplex ELISA platform.

Results Compared with mock infection, VZV infection led to significantly increased levels of the following: interleukin (IL)–8 in all cell lines examined; IL-6 in HBVAFs, HPNCs, and HFLs, with no change in HBVSMCs; and vascular endothelial growth factor A in HBVAFs, HBVSMCs, and HFLs, with a significant decrease in HPNCs. Other cytokines, including IL-2, IL-4, IL-15, IL-16, tumor growth factor– β , eotaxin-1, eotaxin-3, inducible protein–10, monocyte chemoattractant protein–1, and granulocyte macrophage colony-stimulating factor, were also significantly altered upon VZV infection in a cell type–specific manner.

Conclusions VZV infection of vascular cells can directly produce a proinflammatory environment that may potentially lead to prolonged arterial wall inflammation and vasculitis. The VZV-mediated increase in IL-8 and IL-6 is consistent with that seen in the CSF of patients with intracerebral VZV vasculopathy, and the VZV-mediated increase in IL-6 is consistent with the cytokine's elevated levels in temporal arteries and plasma of patients with GCA.

NPub.org/N2/9016a

MRI evaluation of thalamic volume differentiates MS from common mimics

Objective To determine whether MRI evaluation of thalamic volume differentiates multiple sclerosis (MS) from other disorders that cause MRI white matter abnormalities.

Methods There were 40 study participants: 10 participants with MS without additional comorbidities for white matter abnormalities (MS – c); 10 participants with MS with additional comorbidities for white matter abnormalities (MS + c); 10 participants with migraine, MRI white matter abnormalities, and no additional comorbidities for white matter abnormalities (Mig – c); and 10 participants previously incorrectly diagnosed with MS (Misdx). T1-magnetization-prepared rapid gradient-echo and T2-weighted 3D fluid-attenuation inversion recovery sequences were acquired on a Phillips Achieva d-Stream 3T MRI, and scans were randomly ordered and de-identified for a blinded reviewer who performed MRI segmentation using LesionTOADS.

Results Mean normalized thalamic volume differed among the 4 cohorts (analysis of variance, $p = 0.005$) and was smaller in the 20 MS participants compared with the 20 non-MS participants ($p < 0.001$), smaller in MS – c compared with Mig – c ($p = 0.03$), and smaller in MS + c compared with Misdx ($p = 0.006$). The sensitivity and specificity were both 0.75 for diagnosis of MS with a thalamic volume < 0.0077 .

Conclusions MRI volumetric evaluation of the thalamus, but not other deep gray matter structures, differentiated MS from other diseases that cause white matter abnormalities and are often mistaken for MS. Evaluation for thalamic atrophy may improve accuracy for diagnosis of MS as an adjunct to additional radiologic criteria. Thalamic volumetric assessment by MRI in larger cohorts of patients undergoing evaluation for MS is needed, along with the development of automated and easily applied volumetric assessment tools for future clinical application.

Classification of evidence This study provides Class III evidence that MRI evaluation of thalamic volume differentiates MS from other diseases that cause white matter abnormalities.

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