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Abstracts

Articles appearing in the September 2018 issue

GFAPα IgG-associated encephalitis upon daclizumab treatment of MS

Objective To describe a case of glial fibrillary acidic protein α (GFAPα) immunoglobulin G (IgG)–associated encephalitis in a patient referred to us with multiple sclerosis (MS) on daclizumab treatment and to summarize characteristics of 5 additional recent German MS cases of serious encephalitis along with a previously published American case of CNS vasculitis associated with daclizumab.

Methods Evaluation of cause, clinical symptoms, and treatment response.

Results The 6 patients included 4 women and 2 men. The median age at onset was 38 years (range 32–51 years). Clinical presentation was marked by progressing neuropsychologic or neurologic deficits. Additional drug rash with eosinophilia was seen in 3 patients, whereas 2 patients showed a highly active demyelinating process. Examination of CSF samples detected pleocytosis, elevated total protein levels, and GFAPα IgG antibodies, which were not found in serum. In our case, we discovered autoimmune GFAP astrocytopathy associated with encephalitis as secondary autoimmunity, which was steroid-responsive. Clinical outcome of other cases was marked by partial recovery in 4 patients and persistent foster care in 1 patient.

Conclusions Our case of GFAPα IgG-associated encephalitis along with 12 other cases of serious inflammatory brain disorders following daclizumab treatment so far indicates that interfering with natural killer cells and regulatory T cells by anti-CD25 antibody therapy can result in severe secondary CNS autoimmunity in man.

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Gd contrast administration is dispensable in patients with MS without new T2 lesions on follow-up MRI

Objective To assess the diagnostic value of gadolinium (Gd) contrast administration in MRI follow-up examinations of patients with multiple sclerosis (MS) if the T2 lesion load is stable.

Methods We included 100 patients with MS with at least 2 cranial MRI follow-up examinations (mean follow-up time 4.0 ± 2.6 years). MRI was performed at 3T with a standardized protocol including T2-weighted, fluid-attenuated inversion recovery (FLAIR), and T1-weighted contrast-enhanced sequences. Images were analyzed for T2/FLAIR and contrast-enhancing (CE) lesions by 3 independent neuroradiologists. Isolated Gd-enhancing lesions without correlate in T2 and FLAIR images and reactivated Gd^{+} lesions were further assessed for size and signal intensity.

Results We identified a total of 343 new T2 lesions and 152 CE lesions in a total of 559 MRI follow-up examinations. New T2/FLAIR lesions were present in 30% of the scans. Of the Gd-enhancing lesions, 145/152 (95.4%) showed a correlate as a new T2/FLAIR lesion. There were 3 enhancing lesions (1.9% of all enhancing lesions) without T2/FLAIR correlate and 4 lesions (2.6%) that exhibited lesion reactivation or persistent enhancement over time. As a predictive factor of enhancement, we found that enhancing lesions had a higher T2 signal ratio ($T2\ SR_{\text{lesion/normal-appearing white matter}}$: 3.0 ± 0.1 vs 2.2 ± 0.1, $p < 0.001$).

Conclusion The likelihood of missing active lesions is overall small (1.7%) if T2 lesions are stable compared with the previous MRI examination. Lesion reactivation is rare. Our study indicates that Gd contrast administration might be dispensable in follow-up MRI of patients with MS if no new T2/FLAIR lesions and no new neurologic symptoms are present.

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T. Sinnecker, S. Schumacher, K. Mueller, et al. 2016;3:e259. doi.org/10.1212/NXI.0000000000000259

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F. Mir, D. Lee, H. Ray, and S.A. Sadiq. 2014;1:e21. doi.org/10.1212/NXI.0000000000000021