Can we expect long-term clinical improvement through remyelination?

No

Olaf Stuve[1,2,3]

Affiliation:
[1]Department of Neurology and Neurotherapeutics, University of Texas Southwestern Medical Center, Dallas, Texas, USA
[2]Department of Neurology, Klinikum rechts der Isar, Technische Universität München, Germany
[3]Neurology Section, VA North Texas Health Care System, Medical Service, Dallas, TX, U.S.A.

The pathological hallmarks of all multiple sclerosis (MS) subtypes are focal areas or of demyelinating plaques in the central nervous system (CNS), with surrounding inflammation and neurodegeneration. Demyelination leads to decreased nerve conduction velocity, and predisposition of the axons to neurodegeneration due to lack of physical and metabolic support. In fact, axonal loss can be detected at the earliest stages of MS. In addition, accelerated rates of brain atrophy on magnetic resonance imaging (MRI) is evident at all stages of MS in patients with all clinical phenotypes. The pathological substrate of neurological disability is likely the damaged myelin sheet, and, to a larger extend, the loss of intact axons. Damage to axons in the CNS may be acute or subacute, and very likely irreversible. Thus-remyelinating strategies will have very limited efficacy.