Compact Myelin Detachment after metabolic Oligodendrocyte injury

Felix Schweiger¹, Felix Fischbach¹, Julia Nedelcu¹, Friederike Pfeiffer², Uta Chrzanowski¹, Petra Fallier-Becker², Markus Kipp¹

¹ Department of Anatomy II, Ludwig-Maximilians-University of Munich, 80336 Munich, Germany
² Group of Neuron Gila Interaction, Werner Reichardt Centre for Integrative Neuroscience, University of Tübingen, 72072 Tübingen, Germany
³ Institute of Pathology and Neuropathology, University of Tübingen, 72076 Tübingen, Germany

Introduction

Multiple sclerosis (MS) is characterized by demyelination and oligodendrocyte degeneration. The pathology of white matter injury induces neurodegeneration and in consequence accumulation of irreversible clinical disability. Mechanisms leading to oligodendrocyte and myelin degeneration are poorly understood, however centrifugal (early oligodendrocyte cell body pathology) and centripetal (early myelin sheath pathology) mechanisms have been described. Here we investigate structural consequences of Cuprizone-induced centrifugal oligodendrocyte degeneration.

Results

Centrifugal spread of oligodendrocyte damage in cuprizone lesions

Compact Myelin Detachment (CoMyD) at the beginning of active demyelination (i.e., at week three)

CoMyD characteristics

Electrophysiology analysis of corpus callosum axons

Discussion

Our studies show a centrifugal progression of oligodendrocyte pathology during Cuprizone-induced metabolic injury. After initial oligodendrocyte cell body pathology, we propose that retraction of oligodendrocytes’ processes leads to retraction forces, resulting in CoMyD eventually paralleled by axonal injury. Future studies have to show whether CoMyD as well occurs in MS, and which factors regulate this process.

Literature


We would like to thank S. Wübbe, A. Baier, S. Toth and B. Assauer for their excellent technical assistance.

The author states no conflict of interest.