of her brain showed no evidence of any ischemic changes. The splenium of the corpus callosum, the internal capsule and the bilateral occipital subcortical white matter, showed evidence of subacute axonal degeneration that was confirmed by beta-amyloid precursor protein and neurofilament protein. It is important to recognize hypoglycemia per se as a cause of neuroaxonal degeneration. The histological features supporting this diagnosis have been reviewed, along with the special and immunohistochemical staining of this particular abnormality.

**Abstract A14**

**White matter injury: a systematic study**

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The spinal cord injury (SCI) in a rat is a convenient in vivo model to study the massive white matter injury that occurs in stroke, brain trauma, (SCI), etc. The progression of this disease remains poorly understood and the treatments are not effective.

An epidural balloon crush of the caudal thoracic spinal cord caused paraplegia in adult male rats that were maintained for 1 day to 16 weeks post-op and the formalin-fixed spine scanned in 7Tesla Brucker MRI machine. Spinal tissues were decalcified, cut sequentially and stained with LFB + H&E.

The massive SCI results in an extraordinarily long pathological process defined by the severity of inflammatory response and by the unique response by the CNS tissue.

Three overlapping phases can be distinguished:

1. **Acute phase**: initiated by trauma, lasts 3 days, with severe hemorrhages, cellular necrosis and edema in the tissue around the area of injury;
2. **Inflammatory phase**: begins at day 3, with severe infiltration of the site of injury by macrophages, phagocytosis of red blood cells (RBCs), necrotic debris and damaged myelin. The poorly defined site of injury is converted into a cavity of injury (CI) where the inflammation is confined. Edema in the tissue around CI dissipates and there is progressive astroglisis around CI. All tissue debris are internalized by macrophages by day 14 post-op. The macrophage infiltration in contact with leptomeninges becomes infiltrated by fibroblasts and blood vessels starting at day 7 forming a typical granulomatous tissue called arachnoiditis. The areas of CI surrounded by the CNS remain filled with fluid, infiltrated by macrophages that decline in numbers and disappear at 12-16 weeks post-op. CI appears to increase with concurrent irreversible destruction of the surrounding CNS tissue.
3. **Resolution phase**: overlaps with the phase 2, completed at 12-16 weeks post-op, results in elimination of the destructive, macrophage-rich inflammation in CI. Resulting CSF-filled syrinx is surrounded by a wall of severe astroglisis.

Subdural infusion of high doses of dexamethasone for 1-2 week resulted in inhibition but not in elimination of the macrophage infiltration of CI. Long term treatment via direct subdural