

## The progression of kaolin-induced hydrocephalus: light and electron microscopic features in rats

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Hydrocephalus is a common neurological disorder caused by an abnormal accumulation of cerebrospinal fluid within the brain which results in injury to the surrounding brain tissue with neurological deficits. A major factor not usually accounted for is the progressive change over time. In this study, we examined, the changes that occur with time in neurons, glia and neuropil in the brain parenchyma; and ependymal lining of the ventricles in neonatal rats with kaolin-induced hydrocephalus. We induced hydrocephalus in 18 three week-old Wistar rat pups by intracisternal injection of 0.05ml of kaolin solution (250mg/ml in normal saline) while 18 controls had sham injection. The hydrocephalic rats were divided into three groups consisting of six rats each which were sacrificed at one, four and eight weeks post-induction of hydrocephalus along with their age-matched controls. Following sacrifice, 24 of the brain samples were stained with haematoxylin and eosin, cell counts were determined and data analysed using ANOVA at  $\alpha 0.05$ . The remaining 12 were processed for Transmission and Scanning Electron Microscopy (TEM and SEM) and the images analysed descriptively. The laminar organisation of the cerebral cortex was disrupted in all hydrocephalic rats, but neuronal density was significantly increased at 8 weeks ( $127.80 \pm 8.68$  / HPF vs  $85.50 \pm 5.42$  / HPF in controls). An initial denudation observed in the ependymal cell cilia of the ventricular wall was followed by gradual restoration of cilia size and population over time. Ultrastructural changes in the brain parenchyma including enlargement of extracellular space, disruption of intracellular architecture, neuronal degeneration and hydropic changes in cell organelles like the mitochondria were observed with increasing severity as the duration of hydrocephalus increased. Hydrocephalus produces significant structural injury within the brain parenchyma which increases with duration and severity, but there is also evidence of partial structural recovery on the ventricular wall over time.