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ORIGINAL ARTICLE

# Neuronal infection is a major pathogenetic mechanism and cause of fatalities in human acute Nipah virus encephalitis

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Kion Chai Ong and Khong Ying Ng contributed equally to the work.

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## Abstract

### Objectives

Acute Nipah (NIV) encephalitis is characterised by a dual pathogenetic mechanism of neuroglial infection and ischaemia–microinfarction associated with vasculitis-induced thrombotic occlusion. We investigated the contributions of these two mechanisms in fatal cases.

### Materials and methods

We analysed brain tissues (cerebrum, brainstem and cerebellum) from 15 autopsies using light microscopy, immunohistochemistry (IHC), in situ hybridisation and quantitative methods.

### Results

Three types of discrete plaque-like parenchymal lesions were identified: Type 1 with neuroglial IHC positivity for viral antigens and minimal or no necrosis; Type 2 with neuroglial immunopositivity and necrosis; and Type 3 with necrosis but no viral antigens. Most viral antigen/RNA-positive cells were neurons. Cerebral glial immunopositivity was rare, suggesting that microinfarction played a more important role in white matter injury. Type 1 lesions were also detected in the brainstem and cerebellum, but the differences between cerebral cortex and these two regions were not statistically significant.

In the cerebral cortex, Type 1 lesions overwhelmingly predominated, and only 14% Type 1 vs 69% Type 2 lesions were associated with thrombosis. This suggests that neuronal infection as a mechanism of pathogenesis was more important than microinfarction, both in general and in Type 1 lesions in particular. Between the 'early' group (<8-day fever) and the 'late' group (≥8-day fever), there was a decrease of Type 1 and Type 2 lesions with a concomitant increase of Type 3 lesions, suggesting the latter possibly represented late-stage microinfarction and/or neuronal infection.

### Conclusion

Neuronal infection appears to play a more important role than vasculopathy-induced microinfarction in acute NIV encephalitis.

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