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doi: 10.1017/cjn.2019.270

Non-tuberculous mycobacterium encephalitis is rare. Since 2013, a global outbreak of Mycobacterium chimaera infection has been attributed to point-source contamination of heater cooler units used in cardiac surgery. Disseminated M. chimaera infection has presented many unique challenges, including non-specific clinical presentations with delays in diagnosis, and a high mortality rate among predominantly immunocompetent adults. Here, we describe three patients with fatal disseminated Mycobacterium chimaera infection showing initially non-specific, progressively worsening neurocognitive decline, including confusion, delirium, depression and apathy. Autopsy revealed widespread granulomatous encephalitis of the cerebrum, brain stem and spinal cord, along with granulomatous chorioretinitis. Cerebral involvement and differentiation between mycobacterial granulomas and microangiopathic changes can be assessed best on MRI with contrast enhancement. The prognosis of M. chimaera encephalitis appears to be very poor, but might be improved by increased awareness of this new syndrome and timely antimicrobial treatment.

LEARNING OBJECTIVES

This presentation will enable the learner to:

1. Describe the clinical, radiological and neuropathological findings of Mycobacterium chimaera encephalitis
2. Be aware of this rare form of encephalitis, and explain its diagnosis, prognosis and management

Abstract 17

Clinical, neuropathological and molecular features of fatal human pegivirus-associated encephalitis.

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doi: 10.1017/cjn.2019.271

Flaviviruses include many viruses causing encephalitis, including West Nile encephalitis, St. Louis encephalitis, tick-borne encephalitis and Japanese encephalitis. Human pegivirus genotype-1 (HPgV-1) is a lesser known member of the Flaviviridae family and has been identified in human serum, cerebrospinal fluid and brain tissue. Here, we describe two adult patients with fatal HPgV-1-associated encephalitis. Neuroimaging revealed multifocal lesions, initially present in the periventricular and brain stem white matter, then one year later throughout the corona radiata bilaterally with marked involvement of the brainstem and cervical spinal cord. Phylogenetic analyses of HPgV-1 showed clustering of brain-derived sequences from both patients with other human pegiviruses. In both patients, a novel 87-nucleotide deletion in the viral NS2 gene was detected. The presence of positive and negative strand HPgV-1 RNA and viral antigens in both patients indicated viral persistence and replication in the CNS. Autopsy showed lymphocyte infiltration and gliosis predominantly in white matter of the brain and brain stem but, to a lesser extent, also in grey matter. Immunofluorescence revealed HPgV-1 NS5A antigen in lymphocytes as well as in astrocytes and oligodendrocytes. Thus, we hypothesize that the novel deletion in the NS2 coding region may have caused HPgV-1 neuroadaptation or might represent a yet unrecognized genotype of human pegivirus.

LEARNING OBJECTIVES

This presentation will enable the learner to:

1. Describe the clinical and neuropathological features of fatal human pegivirus-associated encephalitis
2. Recognize the importance of molecular analysis in encephalitis cases with unknown etiology

Abstract 18

Absence of age-related neurodegenerative changes during SIV infection and treatment in aged macaques

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doi: 10.1017/cjn.2019.272

The advent of combined antiretroviral therapy (CART) has changed HIV infection from a lethal disease to a chronic infection. CART has substantially mitigated infection-associated immunosuppression, related opportunistic infections and HIV encephalitis, nevertheless a substantial percentage of infected individuals are afflicted with a spectrum of HIV-associated neurological disorders (HAND). As approximately 45% of HIV-infected subjects in developed countries are over the age of 50, it has been hypothesized that infection may exacerbate age related neurodegenerative processes. We used the nonhuman primate SIV infection model to test whether chronic infection of aged primates, with or without CART, is associated with accelerated age-related neurodegeneration. Two dozen aged macaques (average age 18 years at entry 20 years at the end) were divided into two groups, half infected with SICmac251 and the other half not. After 10 months, half of each of these groups were either treated or not with CART and followed for an additional 6 months. We previously reported the clinical and neurobehavioural outcome. Here we compared the molecular and histologic findings in the four groups. Using a broad spectrum of histological markers, we found no evidence in the macaques of neuropathological changes associated with aging in humans. While the number of animals is small and length of infection limited, this study does not support the hypothesis that lentiviral infection or