

# Case Study: Murray Valley Encephalitis (MVE)

Jan Neal

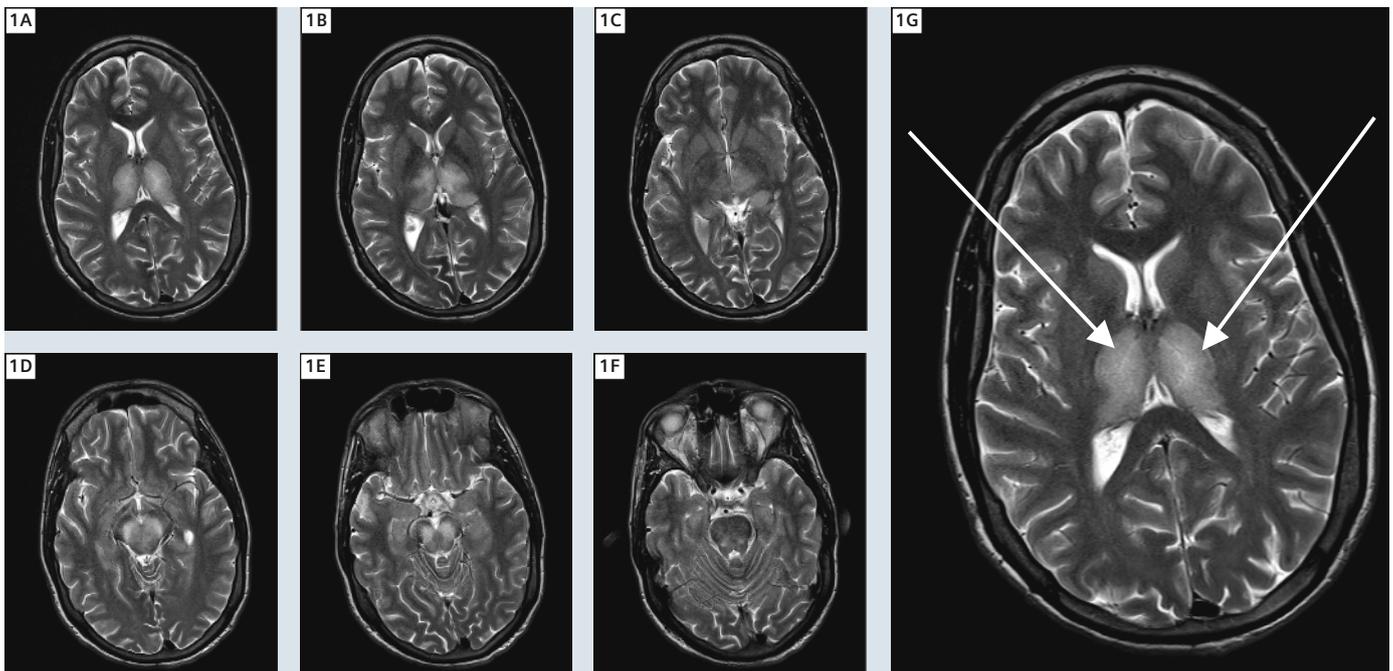
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## Introduction

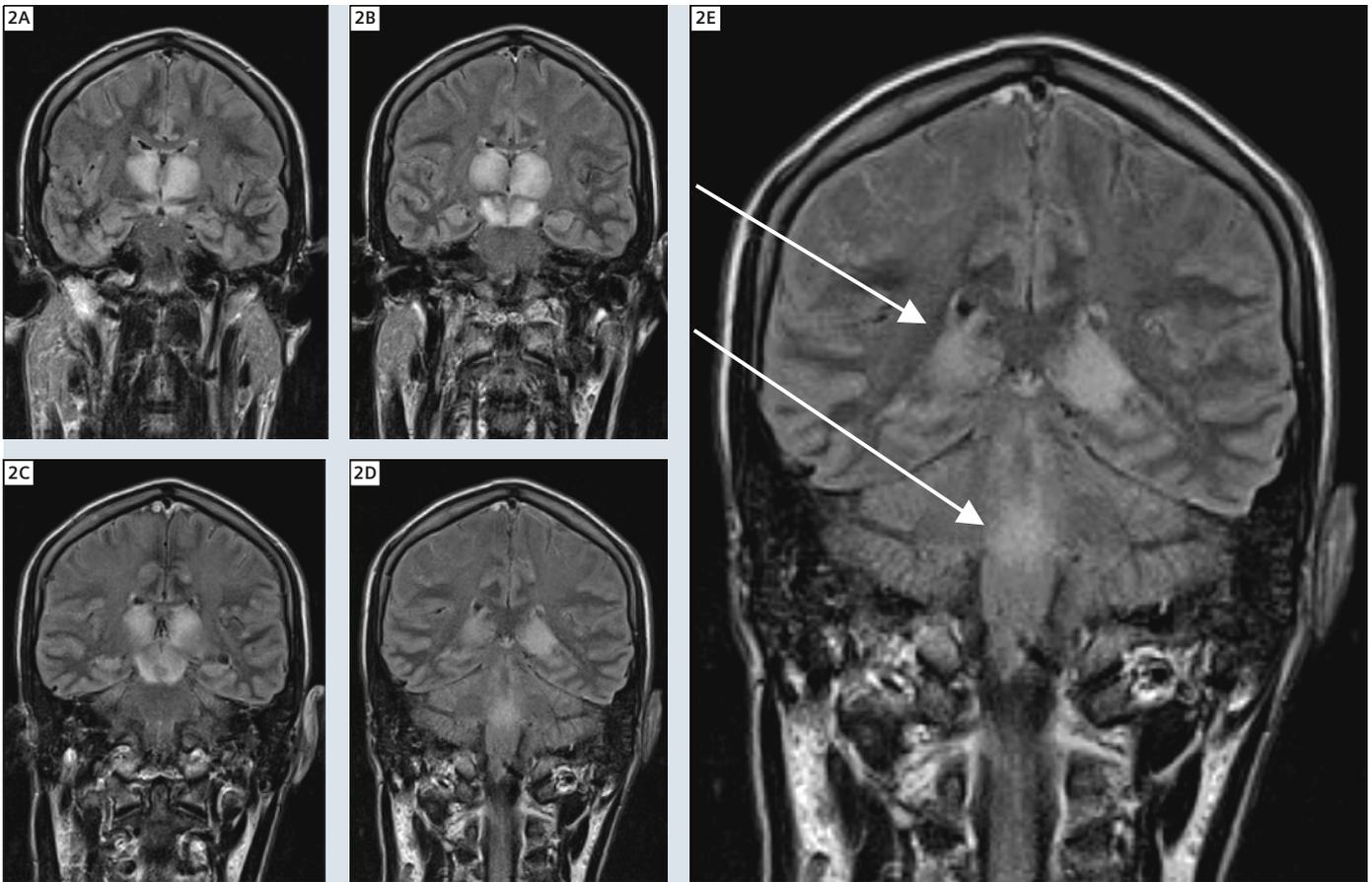
Murray Valley Encephalitis (MVE) is caused by infection with a flavivirus belonging to the Japanese encephalitis (JE) antigenic complex, which also includes St. Louis encephalitis (StLE) and West Nile (WN) virus. The MVE virus occurs in Australia, New Guinea, and probably islands in the eastern part of Indonesia [1]. MVE virus is believed to be maintained in a natural cycle involving water birds and *Culex annulirostris* mosquitos. The natural transmission cycle of the JE group involves infection of a mosquito vector alternating with viral amplification in a variety of

vertebrae hosts. Human disease is incidental to this cycle. Only one in 1,000 to 2,000 infections results in clinical illness resembling JE [1]. They are neurotropic viruses, which cause illness with headache, fever, vomiting followed by drowsiness, mental confusion and in severe cases there may be hyperactive reflexes, spastic paresis, seizures, coma and death. These viruses are becoming increasingly important globally as their geographic ranges steadily increase [2]. MR imaging (MRI) is more sensitive than computed tomography (CT) for detecting

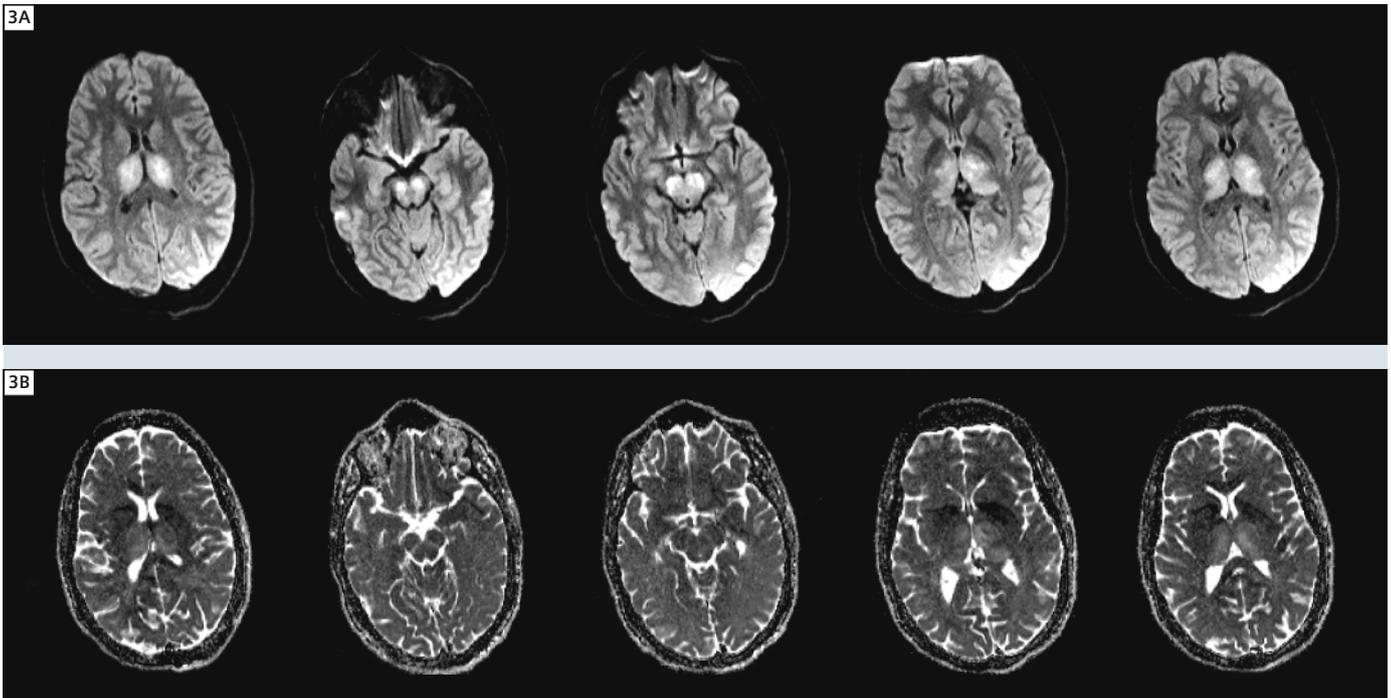
MVE-associated abnormalities such as changes in the thalamus, basal ganglia, mid-brain, pons and medulla. When clinically correlated these changes can be specific to JE but not very sensitive. Mortality among hospitalized patients is about 20–30%. About half of the survivors have residual neurological deficits [2]. There is no vaccine for MVE virus. Prevention relies on mosquito control and avoidance of mosquito bites. There have been no human cases of MVE infection in South Australia for over 30 years.



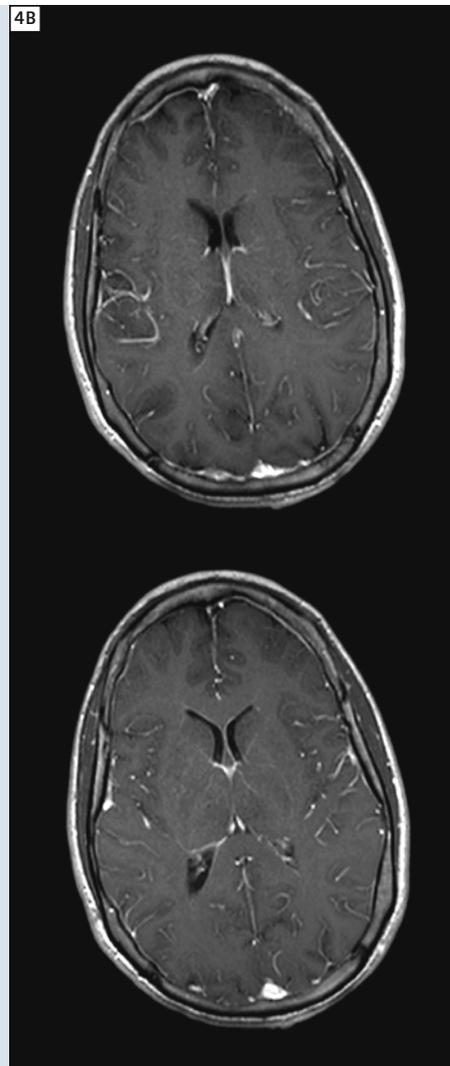
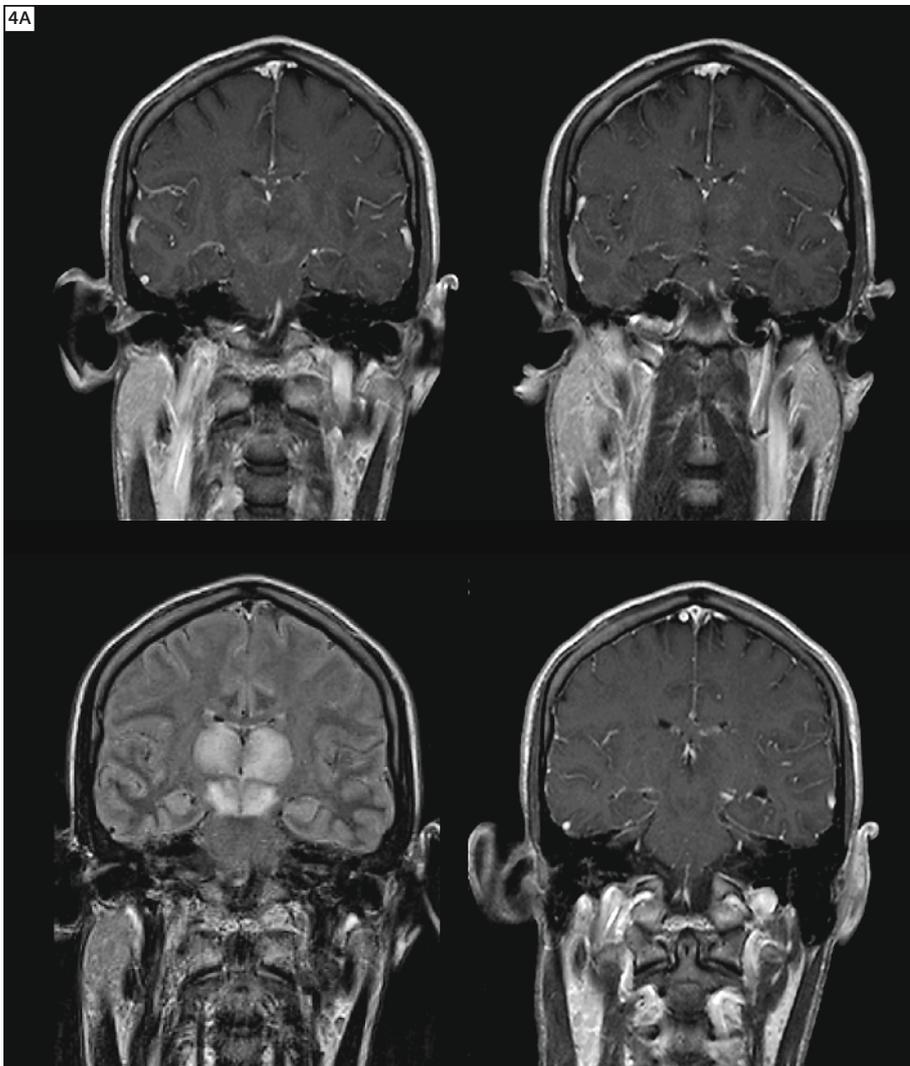
**1** T2-weighted axial images. Abnormal bilateral and symmetric hyperintense signal to central deep grey matter structures including the thalami, substantia nigra, dorsal midbrain and pons.



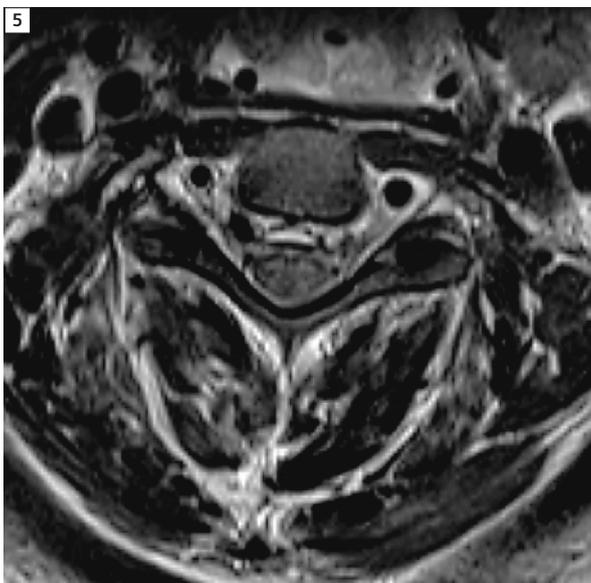
**2** FLAIR coronal images showing abnormal hyperintense signal as seen in Figure 1. T1w images show corresponding hypointense signal.



**3** Diffusion b1000 trace images (3A) and ADC images (3B). These images do not show restricted diffusion. Therefore this is not an acute infarct.



**4** Post contrast imaging, T1w coronal (3<sup>rd</sup> image is a T2w coronal showing the enhancement not evident post contrast) (4A) and T1w axial (4B) showing no enhancement. Pattern, lack of enhancement and the clinical picture do not suggest tumor.



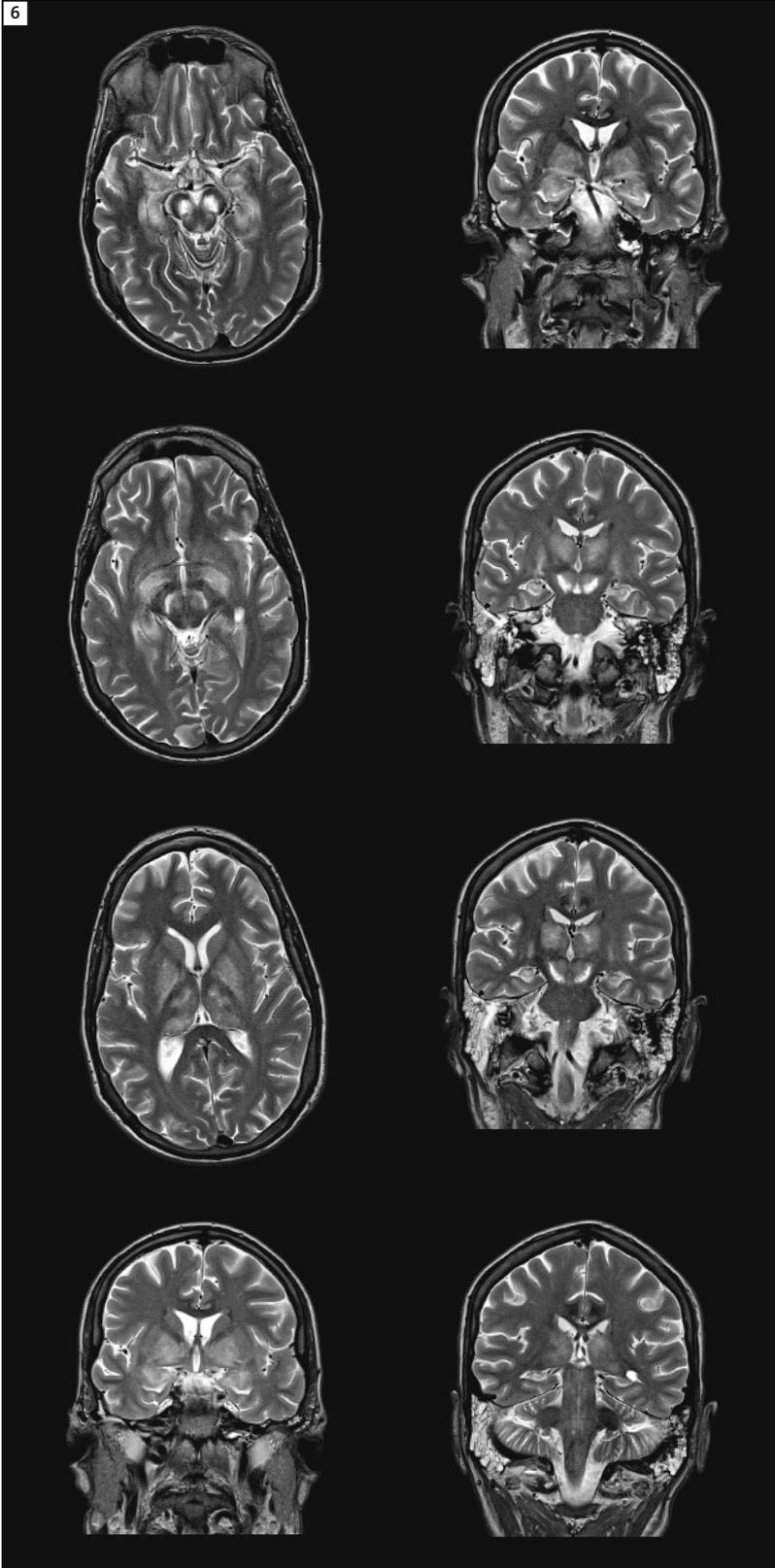
**5** T2w axial image of the cervical spine shows subtle high signal changes involving the anterior portion of the cervical cord.

**Patient history**

A 26-year-old male was admitted to intensive care unit with respiratory failure. MRI head is requested.

**Sequence details**

MRI examination was performed on a Siemens 1.5T MAGNETOM Avanto system, using the head coil. Standard pre and post contrast sequences were acquired. Pre contrast PD/T2-weighted axial, T1w axial, T1w sagittal, FLAIR coronal, diffusion-weighted scans, MR venography and post contrast T1w axial and coronal images were acquired.



**6** After 5 weeks the patient remained ventilated with no signs of neurological improvement. Repeat MRI Brain and c. spine performed. This showed no improvement, there has, in fact, been significant progression of gross abnormal signal intensity with new involvement of the globus pallidus and medial temp lobe.

## Imaging findings

MRI of the brain showed grossly abnormal appearances of the thalami, substantia nigra and dorsal aspect of the brainstem. Given the lack of restricted diffusion and no enhancement a tumor or Herpes Simplex virus (HSV) encephalitis were ruled out. Remainder of the brain was normal. Differential diagnosis would include metabolic processes (osmotic myelinolysis). Correlating to the patient's clinical history it was not felt likely in this case. So an underlying infective aetiology particularly flavivirus infection such as MVE is favored. [3]

## Conclusion

MRI is an excellent clinical tool to aid in the diagnosis of MVE. The classic MR appearance of this virus can assist in expediting the diagnosis. Blood testing must be done in interstate laboratories and can take some time for results to be returned.

### References

- 1 Mackenzie JS, Lindsay MD, Coelen RJ, et al. Arboviruses causing human disease in the Australasian zoogeographic region. *Arch Virol* 1994; 136:447.
- 2 Einsiedel L, Kat E, Ravindran J, et al. MR Findings in Murray Valley Encephalitis. *AJNR Am J Neuro-radiol* 24:1379-1382, August 2003.
- 3 From the radiology report by Dr. S. Knox, Consultant Radiologist, Benson Radiology.



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