Central Australian MVE update, 2001

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The Central Australian public health network became aware of 2 cases of Murray Valley encephalitis during February to March, 2001. Both cases were noted following reports of MVE virus activity in sentinel chicken flocks throughout the Kimberley and Pilbara regions of Western Australia, similar activity in western New South Wales and evidence of sequential north-south spread of virus activity in sentinel chicken flocks throughout the Northern Territory.

Additionally, prior to the detection of human cases of Murray Valley encephalitis, unusually high rainfall had been experienced throughout much of the Northern Territory including in Central Australia, which is commonly referred to as the 'central desert'. Tennant Creek and Alice Springs mosquito monitoring also demonstrated exceedingly high numbers of *Culex annulirostris* mosquitoes well known to transmit MVE virus.

On 22 February 2001 the Centre for Disease Control (CDC), Alice Springs received notification from the Emergency Department of the Alice Springs Hospital, of a suspected case of Murray Valley encephalitis in a 49-year-old female resident of Alice Springs. The second case was notified to CDC, Alice Springs on 3 March 2001, following the diagnosis of Murray Valley encephalitis in a 59-year-old Alice Springs man who had been transferred to the Royal Adelaide Hospital on 28 February 2001. The following are reports of these two cases.

Case 1 first developed symptoms on 19 February 2001. She presented with a 72 hour history of constant frontal headaches and fevers up to 40^{O}C, 48 hours of nausea and vomiting, 24 hours of slurred speech, expressive dysphasia, disorientation and generalised weakness. Within 12 hours of being hospitalised at Alice Springs Hospital the patient demonstrated progressive generalised weakness, respiratory failure and her deteriorating level of consciousness required intubation and ventilation.

Results from serological and cellular investigations of the cerebrospinal fluid (CSF) and serum for Case 1 are listed in the Table. Examination of the initial CSF specimen demonstrated 20 polymorphonuclear cells, 800 monocytes, 20 red blood cells and a raised protein level. Case 1 seroconverted to MVE virus, becoming IgM positive after the initial specimen was collected. A recent infection was supported by the demonstration of rising MVE virus specific IgG titres during follow up. CSF collected from the patient was positive for MVE RNA by polymerase chain reaction (PCR). The patient did not show any serological reactivity to Kunjin virus.

The CSF from Case 1 tested negative for herpes simplex virus, varicella zoster virus, acid fast bacilli, cryptococcus and listeria. Blood cultures were negative and serological testing ruled out infection with Japanese encephalitis, Ross River virus, Barmah Forest virus, Lyme disease or rickettsia.

At 2 months post admission the patient remains in ICU with persistent flaccid quadreparesis requiring ongoing respiratory support. There have been no significant neurological improvements to date.

Case 2 first developed symptoms on 23 February 2001. The patient presented to Alice Springs Hospital on 25 February 2001 with 48 hours of fevers (up to 39.5^{O}C), nausea and vomiting and epigastric abdominal pain. Four episodes of haematemesis (vomiting of blood) were recorded, and the presumptive diagnosis was of a Mallory-Weiss tear.

Over the next 12 hours the patient deteriorated on the ward, where he was found to be delirious with a temperature of 40.5^{O}C. The initial septic screen failed to find a source of sepsis. The patient deteriorated further over the next 24 hours, requiring transfer to a tertiary referral centre. On arrival at the Royal Adelaide Hospital he was noted to have a Glasgow Coma Score of 11, with hypotension, right-sided weakness and right-sided facial droop.

Results from serological and cellular investigations of the CSF and serum for Case 2 are listed in the Table. Initial CSF examination demonstrated 3 polymorphs, 2 red blood cells and 170 monocytes. Case 2 was diagnosed by the presence of MVE virus specific IgM in CSF and serum in combination with a clinical picture compatible with Murray Valley encephalitis. The patient also demonstrated serological reactivity to Kunjin virus. A further convalescent serum sample is required to further investigate whether MVE virus alone, or an infection in conjunction with Kunjin virus was the cause of the patient's encephalitis, although blocking antibody tests suggest that MVE is the likely cause. Insufficient CSF was available for Case 2 for further pathogen testing.

The patient remains in hospital, but is gradually improving. He demonstrates some generalised weakness, but with no specific focal neurological signs.

As of 23 April 2001, there have been no further confirmed cases of Murray Valley encephalitis among Central Australian residents. Falling daily temperatures and dry weather has meant the risk of MVE virus activity has lessened, however, sentinel chickens bled in early April 2001 continued to show MVE activity.

These cases represent the second consecutive year of MVE virus activity and human cases in Central Australia. There has only ever been one documented outbreak of Murray Valley encephalitis in the region prior to the cases demonstrated in 2000. This highlights the need for heightened clinical and public health surveillance in the coming years, particularly in the event of widespread summer rainfall throughout the region.

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Table. CSF and serology results for Murray Valley encephalitis cases acquired in Central Australia, 2001

Case No	Date	Sample	Serum			Serum			CSF		
			MVE HI titre	MVE IgM	MVE IgG-IFA	Kunjin HI titre	Kunjin IgM	MVE PCR	PMN's x10^6/L	Monos x10^6/L	Protein g/L
1	22/2/01	serum	<10	neg	-	<10	neg	neg			
	22/2/01	CSF	-	-	-	-	-	pos			
	22/2/01	CSF							20	800	1.09
	26/2/01	serum	<10	pos	<10	<10	neg				
	22/3/01	serum	<10	pos	640	<10	neg				
2	28/2/01	CSF	-	pos	-	-	-	neg	3	170	-
	01/3/01	serum	80	pos	640	80	equiv				
	14/3/01	serum	80	pos	1280	40	equiv				

CSF cerebrospinal fluid
HI haemagglutination inhibition
PMN polymorphonucleocytes
PCR polymerase chain reaction
Monos monocytes
pos positive
neg negative
equiv equivocal