
Dengue 3 in Cairns: the story so far

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In early December last year, the Tropical Public Health Unit (TPHU) was notified of an adult resident of the Atherton Tablelands who had a positive screening-test for dengue. The patient had a non-specific illness that might have been dengue but he had not recently travelled overseas. Because the screening-test gives a number of false-positive results, it is TPHU policy to delay an investigation in these circumstances while a more reliable test is undertaken in Brisbane.

Before this result became available however, the patient subsequently informed the TPHU that he had had substantial contact with a travellers’ guesthouse on The Esplanade, Cairns North, and several other people at the guesthouse had developed a similar febrile illness. Blood samples were quickly collected from as many of these other individuals as possible and sent to Brisbane for urgent testing; several of these samples tested positive for dengue 3. Because some of these positive people were staff at the guesthouse and had not recently travelled overseas, it was obvious that they had acquired dengue at the guesthouse. Therefore the outbreak was first confirmed on December 12, eight days after the original notification and 16 days after the onset of his symptoms.

Mosquito investigations were commenced immediately. Numerous adult Aedes aegypti were found on the premises including several blood-fed females in the rooms of ill people. The interior of the hostal was sprayed with a commercial pyrethroid aerosol spray. Several pot-plant containers containing Ae. aegypti larvae were also found and emptied. Properties within several hundred metres of the guesthouse were also surveyed, containers emptied and (with permission) interiors sprayed.

Although we were not able to identify the traveller who imported the dengue 3 virus from overseas into Cairns, the virus has a very similar nucleotide sequence to a dengue 3 virus isolated from a traveller who returned to Australia from Thailand in 1993. This suggests that the current virus was imported from southeast Asia.

A travellers’ guesthouse obviously caters to lots of travellers, and we are aware of eight overseas travellers (two of whom contacted TPHU from Spain and Canada via the Internet!) and two interstate visitors who contracted the virus whilst staying in the guesthouse. Because travellers travel, several turned up in other locations in ‘dengue-receptive’ North Queensland whilst still infectious to Ae. aegypti mosquitoes: Innisfail, Mission Beach, Townsville, Magnetic Island and Proserpine. Fortunately no local transmission occurred in these locations, but the inevitable soon happened: spread to other suburbs in Cairns.

In early February TPHU recognised that local transmission of dengue 3 was occurring in Parramatta Park. This is an older, more central suburb with many old Queenslander cottages on small properties. Most of these residences are not screened, and many properties were found to be effectively breeding large numbers of Ae. aegypti mosquitoes in rubbish and garden containers in the backyards. The Parramatta Park outbreak was explosive with 60 confirmed cases occurring in February; a considerable number of these cases were either working from home or unemployed and therefore spending long hours at home exposed to infectious mosquitoes.

Not many travellers stay in Parramatta Park and therefore it was unlikely that the virus would be taken to other travellers’ destinations in North Queensland. However residents of Parramatta Park work, visit and convalesce from dengue in other suburbs of Cairns. Therefore further spread within Cairns was inevitable soon happened: spread to other suburbs in Cairns.

To 25 May 1998, 165 cases have been confirmed. Of these, 31 (19%) have been hospitalised; although some of these only required an overnight stay for IV fluids, two required ICU care. There has been one case of dengue haemorrhagic fever (in an elderly male who fortunately....
only had mild haemorrhage) and one case of dengue encephalopathy (a male in his 20’s who collapsed at work, had several seizures and became increasingly unrousable). Clearly those affected in this outbreak are more ill than those affected in last year’s dengue 2 outbreak in the Torres Strait.

The outbreak seems to be slowing down, and we hope it will soon be over. That the number of cases has been held down to less than 22 so far is a credit to the hard work of the Entomological and Environmental Health staff of TPHU and the Cairns City Council.

A case of infant botulism in South Australia

Rosalind Holland, Communicable Disease Control Branch, South Australian Department of Human Services, PO Box 6, Rundle Mall, South Australia 5000

This report documents the first case of infant botulism recorded in South Australia since 1990.

On 25 May 1998 a case of infant botulism was notified to the Communicable Disease Control Branch. The 6 month old female, from a northern country area of South Australia, was admitted to hospital with paralysis and was diagnosed initially on clinical grounds. She had decreased spontaneous movement, reduced anti-gravity movements, no head movements, and gag and cough reflexes were absent. She was intubated, ventilated and given general supportive treatment.

The baby had become unwell the day before with lethargy and difficulty feeding, and was described by the mother as ‘being uncomfortable’. The mother also reported constipation occurring some days before. The diagnosis was confirmed serologically and by tests in mice.

The baby was breast fed on demand and solid foods had been introduced over the previous 6 weeks. Foods consumed included commercially prepared apricot and rice, pear, mango and apple, and pumpkin either from jars or tins, sweet biscuits and a baby rice cereal. The infant also ate home prepared chicken and vegetable, and toast with Vegemite. No honey or corn syrup was consumed. The family has a dog and keeps chickens although the baby did not have contact with the animals.

Infant botulism results from spore ingestion and subsequent vegetative growth, and in-vivo toxin production in the intestine by Clostridium botulinum. The syndrome affects infants almost exclusively, but can affect adults who have altered gastro-intestinal anatomy and microflora. The illness typically begins with constipation followed by lethargy, listlessness, poor feeding, ptosis, difficulty swallowing, loss of head control, hypotonic extending to generalised weakness (the ‘floppy baby’) and, in some cases respiratory insufficiency and arrest.

There are many sources of spores, including foods and dust. Honey and corn syrup have been implicated in infant botulism. Environment and food sampling in isolated cases, such as the one reported here, is unrewarding because of the ubiquitous nature of the organism.

References


Editorial note

This is the first notification of a case infant botulism in Australia since botulism became a nationally notified disease in 1992. Infant botulism generally occurs between the ages of 2 weeks and 1 year, with 94% of cases occurring at or before the age of 6 months. Clinical severity can range from mild illness with gradual onset to severe respiratory insufficiency and death. Case fatality rates in countries with good paediatric intensive care units are less than 1%. Excretion of C. botulinum toxin and organisms can occur in the faeces for extended periods (weeks to months) but no instance of secondary person-to-person transmission has been documented. An antitoxin is available but is not recommended in the treatment of infant botulism.