A focal, rapidly-controlled outbreak of dengue fever in two suburbs in Townsville, North Queensland, 2001

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Abstract

In April–May 2001 an outbreak of dengue fever occurred in two suburbs in Townsville, north Queensland. This was the first outbreak in the Townsville region since a very large outbreak in 1992–1993. Notification delays resulted in late detection of the outbreak. Once recognised, control measures were implemented and rapid control was achieved. Dengue serotype 2 was the causative virus and 9 cases of dengue fever were documented. The approach to management of dengue fever outbreaks and vector control strategies have been improved and refined in the years since the 1992–1993 outbreak. These measures, in addition to favourable weather conditions, were likely to have contributed to the successful containment of this outbreak. Commun Dis Intell 2002;26:596–600.

Keywords: Aedes aegypti, dengue fever, disease outbreak

Introduction

In 1992–1993 an extensive and prolonged outbreak of dengue fever occurred in Townsville. Control measures appeared to have little impact on the progression of the outbreak and the disease spread to the nearby towns of Charters Towers and Hughenden. By the time the outbreak eventually subsided after 16 months, over 900 cases of dengue fever had been notified to Queensland Health.1 Individual importations of dengue to Townsville (by viraemic travellers) were documented over the following 8 years but no outbreaks were recorded, despite some fairly intense 'risk' periods such as the return of over 2,000 military personnel from dengue-endemic East Timor in February 2000.2

On 15 May 2001 the Tropical Public Health Unit (TPHU) was notified of a case of dengue fever (diagnosed by IgM and IgG seroconversion by enzyme linked immunosorbent assay (EIA)) in a resident of Townsville who had no history of overseas travel. The specimen was referred for confirmatory testing to the Arbovirus Reference Laboratory, Queensland Health Pathology and Scientific Services and initial control measures commenced. Two days later a second IgM positive case was notified to the TPHU. This case likewise had no travel history and the residential address was in the same suburb and in close proximity to the first case. The vector of dengue, Aedes aegypti, is present in north Queensland and is responsible for the spread of the virus from person to person. However, dengue fever is not endemic in north Queensland and transmission only occurs following importation of the virus (via a viraemic human) from a dengue-endemic area. The two reports of locally-acquired cases thus indicated importation of the virus and that subsequent transmission had occurred. This led to an outbreak being declared. This report describes the outbreak and discusses the likely factors that contributed to its rapid control.

Methods

On recognition of a local outbreak of dengue fever, enhanced surveillance for further cases was undertaken. On receipt of a notification of a suspected case, the patient was contacted by TPHU staff to collect information on their movements during their 'exposure' and

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'viraemic' periods. Information on movements during the 'exposure period' (i.e. 3 to 12 days prior to symptom onset) was required to determine where the infection may have been acquired. Details of the 'viraemic period' (one day prior to 12 days after symptom onset) were collected to determine where the person had been whilst infectious and this information was used to inform control measures.

An immediate retrospective investigation was also undertaken at a child day care centre that was situated in close proximity to the houses of the first two notified cases. The aim was to identify and arrange serological testing of those at the centre who had experienced non-specific febrile illness in the previous month. A non-specific febrile illness can be a common presentation of dengue fever in a child. The concern was that if unrecognised cases had occurred in the children at the centre, they could readily have acted as disseminators of virus into the community, as the centre attendees were drawn from many different suburbs of Townsville.

Specific intense control measures focussed on controlling the vector in the vicinity of case houses. The limited flight range of *Aedes aegypti* in the urban environment and its preference for breeding sites that include domestic receptacles such as containers, pot plant bases and roof gutters means premise-to-premise surveys are the appropriate means of control. Surveys were carried out within a 200 metre radius of the residence of the notified case (or at specific premises where cases had spent considerable amounts of time while viraemic), searching for and treating breeding sites if they could not be managed by removal of the source of water. Householders were educated on removal of mosquito breeding sites and were provided with information on individual protective measures. Indoor residual insecticide spraying to ensure ongoing control of adult mosquitoes was undertaken in houses within a 100 metre radius of the residence of cases. Any household members with recent febrile illness identified during these inspections were encouraged to seek medical attention or were followed up to ensure potential cases of dengue fever were not missed.

Broader measures to encourage mosquito control included provision of information to businesses in the outbreak area and media alerts. In addition, cryptic breeding sites such as underground drains and wells were located, inspected and treated.

In an attempt to determine the 'index case' for the outbreak, householders living in the area where the outbreak was first recognised were asked about recent overseas travel to dengue-endemic countries. A laboratory search was also undertaken for patients with a suggestive haematological picture for dengue fever (thrombocytopenia and leukopenia) in the month prior to the onset of illness in the first case.

Initial dengue cases were confirmed by nucleic acid testing or demonstration of a dengue titre fourfold higher than other flavivirus titres by haemagglutination inhibition assay. Once the outbreak was confirmed, any IgM positive result by EIA was classified as a confirmed case.

## Results

The outbreak consisted of a total of 9 cases of dengue fever, occurring in two very distinct waves of transmission (Figure). A case with onset of disease on 30 April 2001 was the first case clinically diagnosed in the outbreak (2 cases with earlier onset dates were only diagnosed retrospectively). Although a General Practitioner (GP) diagnosed dengue fever in this case and also received a positive laboratory report on 9 May 2001, neither GP nor laboratory notified the TPHU immediately and an 8 day delay occurred before the result was received (on 17 May) from the laboratory. A case, notified by a laboratory on 15 May, with onset date of 3 May, was thus the first notification received. At the time of notification, 11 days had elapsed from the time this case first sought medical attention, and 4 days from collection of a blood specimen. The delays meant recognition of the outbreak and implementation of control measures were substantially delayed.

![Figure. Epidemic curve for dengue fever outbreak in Townsville, 2001](image-url)
One staff member and 3 children at the day care centre reported febrile illness during April and early May. The adult and two of the children were tested but results were negative. Recognising that children at the centre with unrecognised or subclinical illness could act as a source of dissemination of virus into the community, extensive measures were taken to prevent infection of children. This included provision of mosquito repellent devices and personal insect repellent for use at the centre. To ensure any possible cases were rapidly identified, parents were provided with information and asked to take their child to a GP immediately if a febrile illness developed.

The two recognised rounds of transmission occurred in late April to early May and again in mid-May (Figure) and involved two suburbs, Mysterton and North Ward. The probable link between these two suburbs was established. Three members of one family group living in Mysterton were infected in the first round of transmission. Following an afternoon visit to the residence of another family member in North Ward on 29 April (on which day all 3 cases would have been viraemic), mosquitoes at that residence were probably infected. A further 2 family members at the North Ward residence and another family member and a visitor in a neighbouring residence subsequently became ill.

As part of the mosquito control efforts, over 420 premises were inspected with 18 (4%) found to have Aedes aegypti breeding sites. Pot plant bases were the most common site of breeding (40% of all sites identified). Many of the residences had backyard wells which were known to be a common breeding site in the previous dengue fever outbreak in Charters Towers. Mosquito larvae or pupae were not present in any wells on inspection but all were treated as a precautionary measure. Internal spraying was conducted in 124 premises which represented 90 per cent of premises in which it was offered.

The outbreak was due to a dengue serotype 2 virus. The index case for the outbreak was not identified. No cases met the criteria for dengue haemorrhagic fever6 however, one patient required hospitalisation with significant haemorrhagic phenomena including haematemesis and epistaxis. The two identified cases in children were relatively mild. Both were diagnosed initially with respiratory illnesses. It was only subsequent to a parent being diagnosed with dengue fever that they were retrospectively tested and their illnesses confirmed as dengue fever.

Laboratory and GP notification delays meant a second round of transmission was inevitable. After recognition of the outbreak, transmission was halted completely. No further cases were notified more than one incubation period after control measures were implemented.

**Discussion**

In contrast to the last outbreak of dengue fever recorded in Townsville during 1992–1993, this outbreak was rapidly controlled. Several factors were likely to have contributed to this outcome. Townsville experienced its coldest May on record in 2001 with a minimum monthly average temperature of 14.4ºC and several minimum recorded temperatures under 10ºC.7 It is well recognised that cooler temperatures can affect the blood feeding activity of Aedes aegypti, prolong the extrinsic incubation period in the mosquito, and reduce adult mosquito longevity. All these factors will reduce the likelihood that transmission of infection will occur. In addition, no rainfall was recorded in Townsville in May.7 The impact of rainfall on dengue transmission is of lesser importance than for other mosquito-borne diseases as the vector predominantly breeds in and around homes in water-filled containers. The lack of rainfall, however, may have limited some potential breeding sites for the mosquito, such as containers in backyards that could collect rainwater.

Weather patterns may have assisted in control of the outbreak but outbreaks in north Queensland have in the past continued through the winter season1,5 and it is unlikely that weather was the single reason for control of the outbreak. Other factors such as overall vector density and viral virulence may also have contributed. Accurate comparisons with the previous outbreak cannot be made. The management of preventive and control aspects of dengue fever has been refined in north Queensland in the last decade. The first 'Dengue Fever Management Plan'8 was written after the extensive dengue fever outbreak in Townsville in 1992–1993. Further important control principles, recognised following a prolonged outbreak in Cairns and Port Douglas in 1997–1999,5 were incorporated into an updated 'Dengue Fever Management Plan for north Queensland, 2000–2005'.9 Implementation of these principles, including well-trained officers responding immediately to the notification of
A history of dengue fever is common among the population of north Queensland as a result of previous outbreaks in the region. Both the 1992–1993 Townsville outbreak and this outbreak were caused by the dengue serotype 2 virus. An epidemic due to another serotype would increase the likelihood of dengue haemorrhagic fever cases. Ongoing vigilance of clinicians and laboratories is required to ensure the risk of dengue fever to the north Queensland population is limited.

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References


An outbreak of Barmah Forest virus disease in Victoria

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Abstract

This report describes the epidemiological and clinical features of an outbreak of 47 cases of laboratory-confirmed Barmah Forest virus disease (BF disease) that occurred in Victoria between January and May 2002. Laboratory-confirmed cases were investigated, and information on travel history and clinical details was collected. Surveillance data from adult mosquito trappings and climatic conditions in the Wellington Shire were also reviewed. The response rate for interviews was 85 per cent (40/47). The most common symptoms reported by cases included arthralgia (95%), lethargy (90%) and maculopapular rash (72.5%). Transmission of BF disease in the Gippsland region was associated with unusually high numbers of Ochlerotatus camptorhynchus mosquitoes. This outbreak was of interest due to the fact that cases of BF disease outnumbered cases of Ross River virus disease (RR disease) in Victoria for the first time since data were available. Similar outbreaks of BF disease, in the absence of RR disease, occurred in Western Australia in 19931 and New South Wales in 1994/1995.2 Although the majority of BF disease cases reported regular outdoor activity during which they could be exposed to mosquito populations, they infrequently take precautions to limit exposure. Further efforts need to be made to educate people of the importance of using repellents and other personal preventative measures. Commun Dis Intell 2002;26:600–604.

Keywords: Barmah Forest virus; surveillance; arbovirus; disease outbreak; Ochlerotatus camptorhynchus

Introduction

Barmah Forest virus (BF) is an alphavirus that was first isolated from mosquitoes trapped in the Barmah Forest of northern Victoria in 1974,3 but was only shown to be pathogenic to humans since 1988.4 BF is the causative agent of Barmah Forest virus disease (BF disease), which is similar to the epidemic polyarthritis caused by Ross River virus (RR).5

Since 1988, BF disease has been reported in Western Australia, Queensland, New South Wales, the Northern Territory and Victoria.6 In Victoria, outbreaks have been previously reported throughout the Murray Valley and the Gippsland areas.7

The Communicable Diseases Section of the Victorian Department of Human Services noticed a greater than expected number of cases of BF disease in February 2002, and this prompted health warnings to be sent out to General Practitioners and media warnings for the Gippsland area, advocating preventative measures to residents and visitors.

This report describes epidemiological and clinical features of the outbreak based on a survey of laboratory-confirmed cases. Adult mosquito surveillance data were also analysed to determine whether there was an association between vector abundance and disease incidence.

References