A review of screening practice identified 2 concerns - the risk of missing latent TB infection (LTBI) due to false negative single-step Mantoux tests in immunosuppressed clients, and the lack of ongoing screening for LTBI in patients who may have further exposure to TB.

A screening algorithm was developed which included a two-step Mantoux test when initial Mantoux results were negative, indications for referral to the TB unit for assessment, and management guidelines for those in whom the initial two-step Mantoux was negative. Additional fields and capacity were requested in SHIP (Sexual Health Information Program) to record serial Mantoux, chest x-ray results and to generate recall lists.

From July 2003 to April 2004, 35 clients (55% of regular attendees to our clinic) have undergone Mantoux testing. Positive results (≥ 5mm induration) were detected in 5/35 (14%) clients – at the first step in 2 (40%), and after the second step in a further 3 (60%). The remaining 30 clients had negative results after the two-step Mantoux test. Of 5 with a positive test, one case of asymptomatic culture-positive pulmonary TB has been detected, and 3 out of 4 clients (75%) with LTBI have commenced preventive treatment.

Currently, ongoing screening for LTBI is thought to be a low priority in HIV management in Australia. These results should stimulate reconsideration of its importance, particularly in other regions with high rates of TB.

This issue will be presented in full at this years CDC Workshop, 12—14 October, Mirambeena Resort, Darwin.

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**An imported case of chikungunya in the Northern Territory and a summary of the ecology of the disease.**

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**Background and ecology based on an extract from “The arboviruses, epidemiology and ecology”**

**Case history**

A 30 year old woman, 18 weeks pregnant with her 4th child presented to the Royal Darwin Hospital emergency department in January 2004 with a febrile illness. She had returned from East Timor 5 days prior where she had been working. The woman had been born in East Timor but had lived most of her life in Australia until taking up a position in Dili.

She described a 6 day history of malaise, fever, headache, back pain and severe myalgias. Arthralgia was not a feature of her illness. An erythematous, maculo-papular pruritic rash had developed in the 24 hours before emergency department review, and coincided with the resolution of fever. Her family had all suffered similar symptoms in the previous weeks in Dili and the woman’s husband had been diagnosed and treated for malaria during this time. Multiple cases of dengue fever had been reported in Dili at the time of her illness.

On examination, the patient was afebrile, alert and cooperative. The rash was widespread, covering the trunk, palms of both hands, arms, back and lower limbs. Cardiorespiratory examination was unremarkable and abdominal examination was notable only for fundal height, consistent with the gestational dates given by the patient. The patient was aware of ongoing foetal movements.

Investigations included 3 consecutive slides for malaria microscopy and *Plasmodium falciparum* antigen testing, which were all negative. The full blood count showed a white cell count of 5.5 x10-9/L with a mild lymphopaenia of 0.9, haemoglobin of 122g/L and platelets of 170x10-9/L. Liver function and renal function were normal and TPPA was non reactive.

A provisional diagnosis of dengue fever was made and the patient returned home with advice to maintain her oral intake and use simple analgesia such as paracetamol when needed.
Dengue fever serology returned days later consistent with past infection only (dengue IgG (EIA) positive, IgM (EIA) negative). Twenty-one days after the onset of illness, the patient’s symptoms had almost completely resolved, apart from persistent malaise. All routine blood tests remained within normal limits; specifically the platelet count did not drop below 170 over the 2 week period of review. A second dengue infection was thought unlikely given the relative mildness of the disease and the lack of any degree of thrombocytopenia but repeat dengue serology was ordered as well as chikungunya virus serology. A positive chikungunya IgM consistent with acute infection was returned (chikungunya IgG HI 320, chikungunya IgM IFA positive). The dengue fever serology was again negative for IgM and a diagnosis of chikungunya was made.

The woman’s pregnancy proceeded to term without complication. She was eventually booked into the labour ward for artificial rupture of membranes, as gestational period had exceeded 44 weeks. A live, healthy infant was delivered spontaneously the next day. She and her daughter were discharged on day 2, post-delivery.

**Chikungunya disease**

*The Virus*

Chikungunya virus, an alpha virus, is one of the 4 species of the Semliki Forest complex, the others being Semliki Forest, Getah, and Mayaro. Chikungunya and o’nyong-nyong viruses are regarded as subtypes of the chikungunya virus species.

*Historical Background*

The word “chikungunya” was first used by the indigenous people of Southern Province, Tanganyika Territory (Tanzania), in reference to a disease which afflicted them in epidemic form in 1952 – 1953. The disease was characterised mainly by a sudden onset of, fever, rash and joint pains. The latter were often severe and sometimes persisted. Chikungunya is a Swahili word meaning “that which bends up” and refers to the stooping posture adopted by patients because of the severity of the joint pains.

The human-biting mosquito *Aedes aegypti* was the suspected vector in the epidemic, as adults were abundant in villages and larvae were abundant in water storage jars. During the epidemic a previously unknown virus was isolated from mosquitoes and humans and its etiological role was confirmed serologically by the demonstration of specific antibodies in recovered patients.

Studies in Africa have uncovered a sylvan transmission cycle between wild primates and *Aedes* mosquitoes of the *Stegomyia* and *Diceromyia* subgenera in the tree canopy of moist forest and semi-arid savannah-woodland.

In West Africa, *Ae. aegypti* was implicated as an urban vector during an outbreak in 1969 in Ibadan, Nigeria, and in 1970 – 1971 in Luanda, Angola. Since 1954, the virus has been identified as the cause of epidemics in the Philippines, Thailand, Kampuchea, India, Sri Lanka, Vietnam, and Burma. In at least some of these outbreaks, *Ae. aegypti* was implicated as the main vector.

Chikungunya virus appears to be enzootic throughout much of tropical Africa, from where it has apparently spread to other parts of the world. There has been a failure so far to find evidence of a feral transmission cycle outside of Africa.

**Disease Associations**

*Humans*

There has been confusion between dengue virus disease and chikungunya virus disease.

Dengue is typically characterised by a fever lasting about 1 week, headache, retro-orbital pain, and backache with generalised body pains and rash. Sometimes there is a diphasic fever pattern and the acute illness can be followed by residual asthenia. The incubation period range is 3-14 days, most commonly 4-7 days.

Chikungunya is a febrile illness characterised by sudden onset, backache, headache, photophobia, arthralgia or arthritis, and rash. The acute illness lasts 3 to 5 days, with recovery in 5-7 days. The incubation period is usually 2-4 days. The most
significant symptom is the joint pain, present in 70% of cases. It may be severe, affecting one joint or several. Reddening and swelling of the joint may occur. The arthritis may persist in a small proportion of cases for months or years and mimic rheumatoid arthritis. The rash, appearing most commonly on the trunk, is macular or maculo-papular, and rarely petechiae may be present. It may be pruritic and occur in short-lived episodes. Chikungunya differs from dengue in that the pain is predominantly located in the joints rather than the muscles, the febrile illness is shorter and usually not diphasic, recovery follows with immunity conferred, and some patients have persistent arthralgia following an acute episode but usually no asthenia.

In contrast with dengue, haemorrhagic manifestations rarely occur in cases of chikungunya and chikungunya should not be listed as a haemorrhagic fever. Severe haemorrhagic symptoms have not been reported in chikungunya cases in Africa. O’nyong-nyong fever has similar symptoms to chikungunya, but o’nyong-nyong may be distinguished by the presence of lymphadenitis which is usually absent in chikungunya patients.

Chikungunya fever can be confused clinically with o’nyong-nyong, dengue, Sindbis, and West Nile infections, so diagnosis should be confirmed by virus isolation as well as serologically. Virus is most readily isolated from the blood within 48 hours of the onset of illness. In Australia, imported or introduced chikungunya is likely to be clinically indistinguishable from the endemic Australian alphaviruses Ross River virus and Barmah Forest virus. Serology tests for these viruses may also exhibit cross-reaction.

**Domestic animals and wildlife**

There are no records of clinical disease in domestic animals or wildlife. After infection with chikungunya, viremia followed by antibody development occurs in Indian rhesus monkeys, African vervet monkeys and baboons. Adult cats, adult fowls, domestic sparrows, and pigeons are refractory to infection by the virus. Wild primates are primary hosts in chikungunya transmission cycles in the wild. In one study, cattle, sheep, goats, horses, and various species of birds showed no viremia after inoculation of virus.

**Epidemiology**

Chikungunya appears to have spread to other parts of the world from Africa to cause pandemics in both the American and Asian tropics. India has had a history of epidemics from 1824 until 1965 when the virus spread to Sri Lanka. Chikungunya became established endemically in Southeast Asia during the late 1950s to early 1960s and was continuously transmitted in the towns and cities in Thailand, Kampuchea, and Vietnam, probably largely by *Ae. aegypti*. In Asia, most recognised outbreaks have been on a large scale in large urban populations with transmission effected by *Ae. aegypti*. In India, it was estimated that during the outbreak in Madras in 1964, nearly 400,000 cases occurred. However, recent evidence indicates that the virus had virtually disappeared from Bangkok by the early 1980s despite abundance of *Ae. aegypti*.

Outbreaks of chikungunya depend upon sufficient rainfall filling the tree-holes or artificial containers preferred for oviposition by the aedine mosquito vectors, resulting in high densities of mosquitoes. Sufficient numbers of non-immune humans must be present to sustain outbreaks.

In urban outbreaks where *Ae. aegypti* is the vector, possibly supplemented by *Ae. albopictus* in Asia, the relation with rainfall pattern has been recorded for several countries. In the 1969 Ibadan, Nigeria outbreak, the frequency of infections increased and decreased parallel with the rainfall pattern. In other areas of South East Asia where rainfall is not markedly seasonal, cases of chikungunya may occur throughout the year.

Where the vector has been the domestic human-biting *Ae. aegypti*, risk to humans has been highest among urban populations, especially those of the lower socioeconomic group, where, in some rural villages, the need to store water is great and the container habitat for mosquito larvae is usually most abundant. Furthermore, the houses occupied by this section of the population are less likely to be mosquito-proof.
Vector and host characteristics

In Asia, virus isolations have been obtained only from *Ae. aegypti*, and it seems certain that this species has been responsible for many of the Asian urban epidemics.

All *Culex* species so far tested including *Cx. quinquefasciatus* have been found refractory to infection with chikungunya virus.

Recent laboratory studies suggest that in South East Asia, *Ae. albopictus* is a more competent vector of chikungunya virus than *Ae. aegypti*.2

Some populations of *Ae. albopictus* have been shown to be better vectors than populations of *Ae. aegypti*, while other populations appear to be less efficient. Hence, *Ae. albopictus* can be regarded as a potential feral vector in India and Thailand, and possibly other countries in South East Asia.

In both Africa and Asia, the virus can doubtless survive for considerable periods, infecting wild primates and moving from locality to locality according to the availability of sufficient numbers of susceptible human hosts.

Prevention and Control

Vaccines have not yet been used to control outbreaks of chikungunya. Control measures should therefore centre around the avoidance of mosquito bites and the reduction in the density of vectors. In urban areas infested by *Ae. aegypti* but free of disease, quarantine measures could be applied to prevent the introduction of virus. Mosquito control may be needed in urban epidemics. The breeding sites of *Ae. aegypti* should be eliminated by reducing the number of water containers, and control of adults and larvae may be necessary. Surveillance of *Ae. aegypti* densities by regular collection of larvae should form the background of any such control program.

Recent Activity

The largest epidemics of chikungunya in recent years have occurred in India and Indonesia.3 The virus is considered to be a relatively recent introduction into South East Asia and an increasing public health problem.3

In Indonesia, chikungunya, first emerged in Bandang, West Java, in December 2002,2 and is spreading eastwards throughout the Indonesian archipelago. It recently spread to East Nusa Tenggara (West Timor) and Central Sulawesi. Hundreds of people in Kupang, the capital of East Nusa Tenggara, were treated at hospitals and public health centres for the disease in February 2003.3

As there is currently no *Ae. aegypti* in the NT except in Tennant Creek, and no *Ae. albopictus* there is little chance of chikungunya cases being contracted in the NT. However cases contracted overseas and imported by overseas travellers can occur in the NT.

In light of the recent cases in West Timor, and the above case from East Timor, there is a potential for more imported cases in Australia. Travellers to Timor in particular, and other areas in the region, should consider mosquito protection while travelling, and advise doctors of their travel history when presenting with similar symptoms after returning.

This recent case of chikungunya and imported cases of dengue illustrates the public health priority to keep the NT free of exotic *Aedes* mosquito vectors such as *Ae. aegypti* and *Ae. albopictus*.

References

Fact sheet

Chikungunya

What is chikungunya?

Chikungunya is a viral infection which causes joint inflammation and general illness. The virus is an arbovirus, of the same family as dengue and o’nyong’nyong.

Chikungunya is sometimes known as Buggy Creek Virus.

Where is it found?

Chikungunya virus is found mostly in South East Asia. It has caused outbreaks in India, Sri Lanka and Thailand in the 1960s but is rarer now. There were some localised outbreaks in Thailand, the Philippines, Sulawesi, Irian Jaya and East Timor in the 1980s. At present, the disease is found in Malaysia, West Java, and particularly East Nusa Tenggara, West Timor. Spread was noted during the rainy season, especially in areas of high rainfall. The disease has not been found in the Northern Territory so far.

How is it spread?

Chikungunya is spread by a bite from an infected *Aedes aegypti* mosquito, which is the same mosquito which carries the dengue virus. This mosquito is found routinely in Cairns and Townsville but in the Northern Territory there is only a localised population in the town area of Tennant Creek. **These mosquitoes are not infected with either dengue or chikungunya.** Chikungunya cannot be spread from person to person.

What are the symptoms?

Symptoms start about 3 to 12 days after infection with the virus. They are flu-like, with fever, chills, and muscular aches. There is pain or inflammation of the small joints of the hands and feet in about 80% of cases. Other symptoms include a sudden severe headache, a flat rash on the arms, legs and trunk, and nausea or vomiting.

The symptoms last for about 3 to 5 days, and if rash occurs, it usually lasts about 2 to 3 days. Sometimes the joint pains can last longer, for more than a month.

These symptoms are very like those of dengue fever so affected people will need to have a blood test done to check for dengue virus.

What is the treatment?

There is no specific treatment for chikungunya fever. Medicines can be given to help relieve the symptoms, such as painkillers.

How can it be controlled?

A vaccine for chikungunya is being researched and there has been some progress in this area. However, there is no vaccine available for widespread use at the moment.

The main way to prevent chikungunya in the NT is by tackling the mosquito that carries the virus.

At the moment, the mosquito is only found in the NT in Tennant Creek. Work is being done by the Medical Entomology Branch and Environmental Health at the moment to get rid of the mosquito from here and to check that it doesn’t spread anywhere else. They also conduct mosquito surveillance in Darwin and at the ports. Sometimes the mosquito can arrive at the coast in fishing boats but these are screened for the mosquito before they land.

Breeding of the mosquito mostly happens during the Wet, but the mosquito eggs survive during the Dry, waiting for the rain. The eggs are laid in still water such as water tanks, drains and pot plant drip trays.
What can be done to reduce mosquito risk?

Reduce breeding areas

- Empty water containers or keep out of the rain. Store empty containers upside down.
- Empty pot plant drip trays, bird baths and pet drinking water once a week and clean thoroughly.
- Screen rainwater and septic tanks and keep covered and sealed.
- Check gutters do not have pooling of water.
- Drain puddles of water.
- Keep fish ponds stocked with fish as they eat the larvae.

Personal protection

- At home:
  - Screen all house doors and windows.
  - Use mosquito coils in enclosed areas.
- When carrying out outdoors activities:
  - Wear loose light coloured clothing, long sleeves and trousers and wear socks.
  - Use an insect repellent, especially ones containing di-ethyltoluamide (DEET) or picardin. Gels or lotions are more effective than sprays.
  - Screen tents.

For more information on mosquitoes and virus ecology contact the Medical Entomology Branch

Darwin 8922 8901

For more information on disease aspects, contact your nearest Centre for Disease Control (CDC)

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