Leptospirosis in the Top End of the Northern Territory: an investigation into the occupational risk to crocodile handlers

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Abstract

The true incidence of leptospirosis in the Top End of the Northern Territory (NT) is unknown, with the reported incidence significantly lower than some other regions of Australia with comparable climatic conditions. The report of a case of leptospirosis in a journalist reporting on crocodile egg harvesting at a fresh water swamp near Darwin, together with 2 other notified cases among crocodile handlers in the 2 preceding years, prompted a serological investigation into the prevalence of previous disease or exposure to the organism among crocodile handlers at a Darwin crocodile park. Serological evidence of exposure was present in 2 of 14 handlers tested. Crocodile handling activities including egg collection are likely to put employees at risk for leptospirosis and rodent control and self-protective measures should be implemented.

Background

Leptospirosis is a zoonotic disease with a global distribution but with an increased incidence in residents of tropical locations and in those exposed by virtue of their occupation. The clinical manifestations are variable, ranging from...
subclinical infection to hepatic and renal failure, severe pulmonary haemorrhage and death. The prevalence of subclinical infection, the absence of pathognomonic features in symptomatic patients, and a lack of awareness of the condition probably contribute to under-reporting of leptospirosis, which is notifiable throughout Australia.

In June 2004 a television journalist reporting on the harvesting of crocodile eggs at a fresh water swamp in rural Darwin was diagnosed with leptospirosis on return to his home state of New South Wales. In the preceding 2 years, 2 cases of leptospirosis (both serovar Hardjo positive) had been documented in crocodile handlers involved in egg collection at the same swamp. Because of their regular exposure to animals and to waterways, it was speculated that leptospirosis may be a specific occupational risk to crocodile handlers. A literature search revealed no other reported cases of leptospirosis among crocodile handlers although there were anecdotal reports accessed via the Internet of the condition arising in a number of alligator handlers in the United States. This paper describes an investigation to assess the risk of leptospirosis to employees involved in crocodile handling and egg collection based at a crocodile and wildlife park.

Methods

With the willing assistance of the management and staff at the park, a delegation from the Centre for Disease Control provided a brief information session about leptospirosis and other infectious occupational risks to workers. Fourteen employees, all present for the information session, consented to serological testing for anti-leptospira antibodies including 1 of the 2 employees who had been diagnosed with serologically proven leptospirosis 2 years previously. Microscopic agglutination testing (MAT) against 21 serovars was performed at the Leptospirosis Reference Laboratory in Brisbane. Specimens with a raised titre to any of the serovars were further tested by enzyme-linked immunosorbent assay (ELISA) for anti-leptospiral IgM. Employees answered a standardised questionnaire about their duration of residency in the Top End, duration of employment at the park, exposure to animals in the park and outside of work, and exposure to the swamp where eggs were periodically harvested. One employee’s questionnaire was not included in the analysis because the answers were incomplete. Statistical analysis was performed by two-tailed independent t-test with an \( \alpha \) value of 0.05.

Results

Of the 14 employees tested, 12 had titres of less than 50 for all 21 of the serovars tested. Of the remaining 2, 1 had a titre of 100 and the other a titre of 200, both for serovar Australis. These 2 serum samples were subsequently tested by ELISA for anti-leptospiral IgM and found to be negative.

The individual with the titre of 100, who had been an employee for 7 years, reported an undiagnosed febrile illness several years previously with onset 1 week after a trip to the swamp to collect crocodile eggs. He reported that several other members on the trip also became ill afterwards with similar symptoms of fever, headaches and muscle aches. The other members on the trip were not available for testing as they were no longer employees of the park.

The other individual with a raised titre reported that he had been diagnosed with acute leptospirosis while working as a dairy farmer in New South Wales in the 1970s and had not had any illnesses consistent with acute leptospirosis since commencement of employment at the park 5 years previously.

One employee who had had serologically proven leptospirosis 12 months previously (with a MAT titre of 1600 to serovar Hardjo) subsequently had a titre of less than 50 to serovar Hardjo and to the other 20 serovars tested. All respondents reported that they had regular occupational exposure to animals and to waterways.

The mean duration of employment at the park for the seronegative employees was 2.4 years, and for the 2 seropositive employees it was 6.4 years (two-tailed independent t-test, \( p=0.19 \)).

The mean duration of residency in the Top End for the seronegative employees was 8.2 years, and in the 2 seropositive employees it was 6.4 years (two-tailed independent t-test, \( p=0.83 \)).
Discussion

Global interest in leptospirosis has increased in recent years following reports of large scale outbreaks of the disease especially, but not exclusively, in developing countries. Infection occurs in temperate regions predominantly by occupational exposure, by occupational or non-occupational exposure in endemic tropical regions and also in the form of outbreaks as have occurred in parts of the world which experience periodic flooding. Endemicity in tropical regions is thought to be promoted by the ability of leptospires to survive in warm and moist environments and by the expanded biomass of infected animals living in proximity to human populations.

Leptospira are obligate aerobic spirochetes that live either within animal hosts (particularly within renal tubules) or freely in the environment. The genus is divided into genomospecies on the basis of genetic homology, but more commonly leptospira are still referred to with respect to their reaction with antisera directed against their lipopolysaccharide capsules. While leptospires can occasionally be demonstrated by dark-field or immunofluorescent microscopy, and techniques for cultural isolation and antigen detection exist, most cases of acute leptospirosis are diagnosed serologically. Other serological methods exist, however, microscopic agglutination testing (MAT) remains the gold standard test. The test is difficult to perform and interpretation can be complicated. The MAT is able to determine the infecting serovar or closely antigenically related serovars. Where the serovars representative for a region are known and included in the antigen panel, the MAT can be used to accurately determine the infecting serovar. Convalescent phase titres are more reflective of the infecting serovar. Cross reaction of serovars, especially in the acute phase and within the same serogroup, is common and paradoxically the titres of a non-implicated serovar may be highest initially owing to shared antigens between the leptospires and rapid anamnestic responses to those serovars to which the individual has been previously exposed. A fourfold rise between acute and convalescent titre is generally accepted as confirmation of acute disease although a single titre as low as 200 in a patient with a compatible illness defines a probable case under US Centre for Disease Control criteria. Australian guidelines define that a single titre of ≥ 400 with a positive ELISA IgM result constitutes a confirmed case. Titres generally take months or years to fall to low levels. MAT has been recommended as the most appropriate test for serological surveys for leptospirosis, and a titre of ≥ 100 is generally considered evidence of past exposure.

Human infection generally occurs by exposure to water or soil contaminated by the urine of infected animals. The usual mechanism is that the leptospires in contaminated water or soil gain entry through abrasions in the skin or through mucous membranes but direct transmission from animals also occurs. Since the identification of miners and sewer workers as occupational risk groups early last century, numerous other occupational risk groups have been identified. Farmers in a number of agricultural industries account for much of the occupational risk to leptospirosis globally and rice workers are known to have been affected since ancient times. In Australia, banana farmers and cane cutters are clearly identified at-risk occupations and have been specifically targeted for risk-minimisation strategies. Rodent control measures have likely contributed greatly to the reduction of disease in these industries. These industries have also been implicated in disease outbreaks in other countries. Smith and colleagues demonstrated that the acidic soil of cane fields which were experimentally contaminated by the urine of infected rodents could sustain viable leptospira for several weeks.

Workers at various levels of the beef and dairy industries are also well-defined at-risk groups throughout Australia as they are throughout the world where serovars Hardjo and Pomona are commonly implicated. Leptospirosis is often asymptomatic in cattle but it is also implicated in symptomatic infections including bovine abortion.

There are several crocodile farms and parks in the NT and Queensland. Most of these parks and farms are tourist attractions and some are also engaged in research activities or in harvesting for meat and leather products. Some
parks periodically harvest eggs from the crocodile’s natural habitat either in rivers or swamp areas. There are no reports of direct transmission of leptospirosis from crocodiles to humans and crocodile handling in Australia has not been previously established as an at-risk occupation. Crocodiles in Australia have not been investigated for carriage of leptospires though the related crocodilian caimans from South America have been shown to be seropositive for anti-leptospiral antibodies.

In Australia, the most commonly reported serovars are Hardjo, Zanoni and Australis which were responsible for 64.1% and 60.5% of reported infections in 2002 and 2003 respectively. The overall rates of notification in Australia ranged from 0.83 to 1.35 per 100,000 for the years 2000 to 2002 with the highest reported rate coming from the major banana growing region of Innisfail, Queensland which had a rate of 119.7 per 100,000 population. Most cases in Australia report exposure to animals, the most frequently implicated of which are rats, cattle and mice. Exposure to dogs is also very commonly reported but their importance to the epidemiology of leptospirosis remains unclear.

The serovar Australis accounted for 11.3 to 18.8% of reported infections in the years 2000 to 2002 and is mainly found in the NT and coastal north-eastern Australia down to the rainforest areas of northern New South Wales. The main carriers of the serovar are thought to be rats and native marsupials.

In the NT, in the 6 years 1998 to 2003 (see Table), 27 cases of leptospirosis were identified, equating to a mean annual incidence of 2.25 per 100,000 population for this period. Residence of 26 of the 27 cases were from the Top End of the NT with the remaining case giving both Top End and Tennant Creek area addresses. Of the 27 cases, 15 (55.5%) were due to the serovar Australis. The majority of cases (18 of 27) occurred in the wet season months November to April. Employees of the dairy or beef industries accounted for 8 of the 27 cases. The other occupations recorded included 5 who worked on or in the water (a tugboat operator, a river worker, a professional mud crabber and 2 crocodile handlers involved in harvesting crocodile eggs), 2 tradespeople/labourers, 3 unemployed persons, 1 each in a national park officer, a policeman and a clerk, and 4 employed in home duties. In only 2 of the 27 reported cases was no occupation recorded.

In late 2000, a cluster of 6 cases of leptospirosis (serovar Australis) were reported in the Top End. Of these 6 cases, 4 were in a group of women who had been gathering turtle eggs while the other 2 cases occurred in recreational duck hunters. Although records are not complete, an occupational or recreational risk factor was identifiable in 22 of 27 cases from 1998 to 2003. The presence of case clustering and the predominance of several serovars suggest that infections may not occur because of widespread soil contamination with leptospires in the Top End as occurs in some endemic tropical regions. Clusters of environmental acquisition of leptospirosis however still occur in small outbreaks. The difference between the incidence of leptospirosis reported in the Top End and that reported in specific regions in tropical and subtropical Queensland might best be explained by the absence in the Top End of the large scale banana and cane industries which account for much of the incidence in Queensland.

**Notified leptospirosis cases from the NT 1998-2003.**

The full extent of exposure to leptospira in the NT, particularly the Top End, remains unknown. In endemic regions, the rate of subclinical infection is reported to be relatively high. Many patients may also present with undifferentiated febrile illnesses. These reasons, along with a lack of awareness of the condition, may contribute to under reporting of the condition. Notification of 3 cases of acute leptospirosis following exposure to egg harvesting by crocodile handlers at a swamp, together with the finding of 2 more employees with positive serology for leptospirosis, suggests that this condition may pose an important risk to crocodile handlers. The negative IgM antibody titres for both workers suggest that neither were acutely infected and that exposure occurred at some stage in the past. It is unclear whether the worker who had had leptospirosis diagnosed almost 30 years ago is likely to have had persistently elevated titres from that episode, or whether the raised antibody levels are more likely to represent more recent exposure. It is
noteworthy that the titre for 1 employee with documented leptospirosis 12 months previously was already less than 50 on the repeat sample, highlighting that even in documented infections representative antibody levels may wane within months.

It is possible that leptospires are transmitted directly from infected crocodiles to the hands of handlers or that handlers may be indirectly exposed by water contaminated with the urine of leptospiruric crocodiles. It is probably more likely that handlers are exposed because the

<table>
<thead>
<tr>
<th>Date diagnosed</th>
<th>Sex</th>
<th>Age</th>
<th>Occupation / Recreation risk factor</th>
<th>Serovar</th>
<th>MAT titre</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/2/98</td>
<td>M</td>
<td>29</td>
<td>National park officer / ?</td>
<td>Australis</td>
<td>1600</td>
</tr>
<tr>
<td>24/3/98</td>
<td>M</td>
<td>38</td>
<td>Cattle worker</td>
<td>Tarassovi</td>
<td>800</td>
</tr>
<tr>
<td>22/7/98</td>
<td>M</td>
<td>35</td>
<td>Cattle worker</td>
<td>Hardjo</td>
<td>3200</td>
</tr>
<tr>
<td>11/3/99</td>
<td>M</td>
<td>26</td>
<td>Cattle worker</td>
<td>Hardjo</td>
<td>6400</td>
</tr>
<tr>
<td>15/3/00</td>
<td>M</td>
<td>23</td>
<td>Cattle worker</td>
<td>Hardjo</td>
<td>6400</td>
</tr>
<tr>
<td>29/3/00</td>
<td>M</td>
<td>36</td>
<td>Cattle worker</td>
<td>Hardjo</td>
<td>1600</td>
</tr>
<tr>
<td>17/4/00</td>
<td>M</td>
<td>19</td>
<td>Cattle worker</td>
<td>Hardjo</td>
<td>800</td>
</tr>
<tr>
<td>4/9/00</td>
<td>M</td>
<td>43</td>
<td>Abattoir worker</td>
<td>Pamona</td>
<td>3200</td>
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<tr>
<td>3/11/00</td>
<td>M</td>
<td>45</td>
<td>Tug boat worker / Duck hunting</td>
<td>Australis</td>
<td>6400</td>
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<tr>
<td>23/11/00</td>
<td>M</td>
<td>57</td>
<td>? / Duck hunting</td>
<td>Australis</td>
<td>&gt;6400</td>
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<tr>
<td>14/12/00</td>
<td>F</td>
<td>55</td>
<td>Home duties / turtle hunting &amp; egg gathering</td>
<td>Australis</td>
<td>&gt;6400</td>
</tr>
<tr>
<td>14/12/00</td>
<td>F</td>
<td>56</td>
<td>Home duties / turtle hunting &amp; egg gathering</td>
<td>Australis</td>
<td>800</td>
</tr>
<tr>
<td>21/12/00</td>
<td>F</td>
<td>46</td>
<td>Home duties / turtle hunting &amp; egg gathering</td>
<td>Australis</td>
<td>6400</td>
</tr>
<tr>
<td>21/12/00</td>
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<td>50</td>
<td>Home duties / turtle hunting &amp; egg gathering</td>
<td>Australis</td>
<td>6400</td>
</tr>
<tr>
<td>25/5/01</td>
<td>F</td>
<td>22</td>
<td>? / ?</td>
<td>Hardjo</td>
<td>3200</td>
</tr>
<tr>
<td>15/10/01</td>
<td>M</td>
<td>32</td>
<td>Labourer / ?</td>
<td>Australis</td>
<td>1600</td>
</tr>
<tr>
<td>22/10/01</td>
<td>M</td>
<td>61</td>
<td>Clerical duties / ?</td>
<td>Australis</td>
<td>3200</td>
</tr>
<tr>
<td>1/11/01</td>
<td>M</td>
<td>21</td>
<td>Unemployed / Swamp walking</td>
<td>Australis</td>
<td>800</td>
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<tr>
<td>30/1/02</td>
<td>M</td>
<td>41</td>
<td>Worker on a river</td>
<td>Australis</td>
<td>400</td>
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<tr>
<td>20/5/02</td>
<td>M</td>
<td>44</td>
<td>Crocodile handler</td>
<td>Hardjo</td>
<td>&gt;6400</td>
</tr>
<tr>
<td>16/10/02</td>
<td>M</td>
<td>60</td>
<td>Tiler / Goose hunting</td>
<td>Australis</td>
<td>&gt;6400</td>
</tr>
<tr>
<td>16/12/02</td>
<td>M</td>
<td>43</td>
<td>Unemployed / Rural living, barefoot with sores in Indonesia with cattle, goats and rats</td>
<td>Cynopteri</td>
<td>400</td>
</tr>
<tr>
<td>5/6/03</td>
<td>M</td>
<td>55</td>
<td>Crocodile handler</td>
<td>Hardjo*</td>
<td>1600</td>
</tr>
<tr>
<td>18/9/03</td>
<td>M</td>
<td>50</td>
<td>Unemployed / Pig hunting</td>
<td>Zanoni</td>
<td></td>
</tr>
<tr>
<td>4/11/03</td>
<td>M</td>
<td>46</td>
<td>Professional mud crabber / Goose hunting and fishing</td>
<td>Australis</td>
<td>&gt;6400</td>
</tr>
<tr>
<td>6/11/03</td>
<td>M</td>
<td>32</td>
<td>Policeman / ?</td>
<td>Australis</td>
<td>&gt;6400</td>
</tr>
</tbody>
</table>

*Also has an unchanging serovar of Australis, MAT titre 200 suggesting possible past infection

Table: Cases of leptospirosis in the NT, 1998—2003

It is possible that leptospires are transmitted directly from infected crocodiles to the hands of handlers or that handlers may be indirectly exposed by water contaminated with the urine of leptospiruric crocodiles. It is probably more likely that handlers are exposed because the swamps and waterways from which eggs are harvested are contaminated by infected urine of rodents or other animals. If more cases of leptospirosis are documented in this occupational group, a serological survey of anti-leptospiral antibodies in crocodiles may be warranted to elucidate this mechanism. Without a serological survey of Top End residents to establish a baseline incidence of seropositivity for anti-leptospiral antibodies, it is not possible to definitively conclude that seropositivity is higher in the crocodile handling group.
Conclusion

Notifications of leptospirosis in the Top End suggest that many cases occur in small clusters when individuals are exposed by virtue of their employment or by recreational activities. There have also been some unclustered cases with no occupational or recreational risk factors identified, though incomplete data capture is acknowledged. Environmental acquisition probably accounts for many cases and these tend to occur in small clusters. While the Top End lacks some of the agricultural industries responsible for much of the incidence of leptospirosis in Australia, anecdotal evidence suggests that crocodile handling may be a risk factor for this condition and the finding of raised titres in 2 of 14 workers supports this but does not prove it conclusively. The findings are sufficient to support the promotion of rodent control measures and self protective measures for crocodile handlers and others employed in the Top End who are regularly exposed to muddy or watery environments. While the Top End lacks some of the agricultural industries responsible for much of the incidence of leptospirosis in Australia, anecdotal evidence suggests that crocodile handling may be a risk factor for this condition and the finding of raised titres in 2 of 14 workers supports this but does not prove it conclusively. The findings are sufficient to support the promotion of rodent control measures and self protective measures for crocodile handlers and others employed in the Top End who are regularly exposed to muddy or watery environments. Clinicians in the Top End should be aware of the types of occupational and recreational activities that predispose to this condition and should have a low threshold for testing for leptospirosis in those with consistent symptoms.

Tom Snelling was the principle author who assisted with formulation of the study design, contributed principally to the on-site visit and who performed the literature search and prepared the manuscript. Vicki Krause supervised the project, assisted with the on-site visit, assisted with the study design and updated/quality assured identified leptospirosis cases. Karen Dempsey assisted with the on-site visit including the collection of blood samples and reviewed the manuscript. Lee Smythe provided expert advice on leptospirosis, advised on technical aspects of the serological testing and reviewed the manuscript. Meegan Symonds and Michael Dohnt performed the serological testing.


References

Editorial- Leptospirosis in the Northern Territory...Hunters beware!

In writing the above article much effort was put into “quality assuring” the past leptospirosis notification data to make it as complete as possible regarding cases diagnosed in the Northern Territory (NT) and documenting their identified serovars. Requests regarding laboratory diagnoses of cases of leptospirosis were made to laboratories routinely serving the NT over the years, as well as to the WHO/FAO/OIE Collaborating Centre for Reference and Research on Leptospirosis in Brisbane Queensland. This review was productive – with most missed cases or serovar results captured via ‘cross-checking’ with the Collaborating Centre in Queensland.

So, to start off with, there are a few updates or corrections following the report in the last Bulletin* of the “first” case in the NT (from June 2004) of leptospirosis due to Leptosira Tarassovi. As noted in the above article’s Table, L. Tarassov, was identified, as the causative agent in a 1998 case (formerly listed as an unknown serovar), a cattlemann/ stockman working in the Katherine, NT area. Looking also at the year before, 1997 (not listed in the Table) another case of L Tarassovi was identified (source, updated NT Notifiable Diseases System), in a meatworker, also from the Katherine area. From 1998 onward, 3 additional cases of previously un-notified leptospirosis were found, 2 in 2000 (both serovar Hardjo) and 1 in 2002 (serovar Cynopteri, common in Indonesia). A previously unknown serovar from 2000 was also found to be Hardjo.

Although no further cases for notification were detected during the 6 year period, 1992 to 1997, only 5 cases were notified, whereas for the following 6 year period, 1998 to 2003, 27 cases were notified. While missed notifications in the earlier 6 year period are possible, there is a clear trend to increasing numbers of cases of leptospirosis being diagnosed in the NT, consistent with a global trend.

The work by Snelling et al set out to determine whether crocodile handlers were at increased risk of leptospirosis due to their occupation – and while this remains unclear – recreational hunting (duck, goose, turtle and pig) appears to be a well represented past-time for those acquiring leptospirosis in the Top End. Hunters in the NT need to be aware of the risk of exposure when tramping though swamps and along waterways and institute personal protection measures and know to seek medical attention for symptoms suggestive of leptospirosis. Symptoms occur in the range of 4 to 19 days after “at-risk” exposure activity and often include sudden onset of fever, headache, chills, severe muscle pain (in calves and thighs) and bloodshot eyes. Further manifestation may include those of meningitis, bi-phasic fever, rash, hepatorenal failure, haemorrhage, cardiac and pulmonary involvement and mental confusion.

Reference


Acute post-streptococcal glomerulonephritis in a remote community

Lesley Scott, CDC, Darwin

Acute post-streptococcal glomerulonephritis (APSGN) was notified in 2 children from a remote Aboriginal community in September 2004. As these children were not from the same household this met the definition1 for prompting a community screen as a public health response and therefore all individuals from the households, as well as all children up to the age of 15 years were recommended to be screened.

The household contacts were screened for scabies, skin sores, facial and peripheral oedema, haematuria, and hypertension.

All contacts aged 3 – 15 years were treated with IMI benzathine penicillin regardless of whether they had skin sores. Other household contacts were only treated if they had skin sores present.
Table 1. Results of the household contact tracing

<table>
<thead>
<tr>
<th>Scabies</th>
<th>Skin sores</th>
<th>Oedema</th>
<th>Haematuria (= Urine dipstick positive for blood)</th>
<th>BP elevated</th>
<th>Penicillin</th>
<th>Total screened</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>14</td>
<td>0</td>
<td>7</td>
<td>0</td>
<td>20</td>
<td>33</td>
</tr>
</tbody>
</table>

Table 2. Results of the community screen

<table>
<thead>
<tr>
<th>Scabies</th>
<th>Sores</th>
<th>Oedema</th>
<th>5% permethrin</th>
<th>Penicillin</th>
<th>Total screened</th>
</tr>
</thead>
<tbody>
<tr>
<td>33</td>
<td>98</td>
<td>0</td>
<td>24</td>
<td>75</td>
<td>300</td>
</tr>
</tbody>
</table>

Of the 37 household contacts 33 were screened (a mother refused assessment of her children advising that they did not have scabies, skin sores or oedema). There were 17 children aged between 3 and 15 years and 3 adults with skin sores who were all treated with benzathine penicillin (Table 1).

Community screening of those up to 15 years of age was commenced. The health centre staff identified, via a ‘community population list’, a possible 850 children in this age group. Posters about the screening were displayed around the community and the local health staff advised community groups. A team consisting of 2 community staff and 3 Centre for Disease Control (CDC) staff commenced the screening. A community list was used to identify the children and keep track of those screened. Each child was screened for scabies, skin sores, facial and peripheral oedema and offered treatment with 5% permethrin cream and/or benzathine penicillin as required. The team had 2 members in the community going from house-to-house assessing the children with another member transporting children to the clinic where the remaining staff were administering the benzathine penicillin.

Many children were away from the community due to ceremonies. After an initial 3 days of screening, assistance was offered from the child health team (1 nurse) and another nurse from CDC continued the screening for a further 2 days. At the end of this time all readily available children (n = 300) had been screened and treated (Table 2). No new cases of APSGN were identified. This is somewhat unusual as past community screening has usually identified further cases.

Although only 318 children (including those under 15 years in the household group) were screened the team had completed the house-to-house visiting of all those that were at home. Many families attended the clinic without prompting after seeing information on the posters.

Most parents were aware of the reason for screening and were wanting their children’s skin sores treated.

Surrounding communities were advised to be alert for further cases of APSGN. Subsequently 2 probable cases were identified, each from a different community and as yet these are unconfirmed.

All medical staff are encouraged to notify CDC of suspected cases of APSGN to enable early public health interventions to be put in place.

References

Barge Belly

A gastrointestinal illness outbreak on a barge at sea.

Philippa Binns, CDC Darwin and Master of Applied Epidemiology Program, NCEPH, ANU, Canberra; Karen Dempsey, OzFoodNet enteric disease epidemiologist, CDC Darwin; Leah Campbell, environmental health officer, CDC, Darwin; G.M. (Mike) Galvin, Maritime Medical Officer, Claremont, WA.

Background

On Thursday 22 July 2004 the Centre for Disease Control (CDC) at the Northern Territory Department of Health and Community Services in Darwin was alerted by an offshore engineering and construction company of a gastrointestinal illness outbreak on board a barge in the Timor Sea being towed from Singapore to Darwin. It was reported that about half of the 150 crew had experienced diarrhoea during the previous 2 weeks, with 1 person being diagnosed, on leaving the barge, with amoebic dysentery and salmonellosis. The doctor on board had instituted food and hygiene interventions. There had been no further cases that day and only a very few in the previous days suggesting that the peak of the outbreak had passed. Immediate investigation at sea by CDC, was not considered necessary. However on arrival in Darwin Harbour on Monday 26 July 2004 there were further cases. A site visit was planned for the following day.

Methods

On Tuesday 27 July the CDC outbreak team consisting of an enteric disease epidemiologist, environmental health officer, epidemiology registrar and medical student visited the barge. Two meetings were held on board the barge. The first was with the barge medical officer, administration manager, occupational health and safety officer and the barge superintendent and the second with union representatives. A detailed history of the events and sequence of the outbreak was documented, with the barge medical officer providing most of the information. No questionnaires were administered due to the long period since initial cases and the marked decline in current cases. At these meetings the initial recommendations for preventing further cases on the barge were provided. The probability of secondary transmission to families from crew members returning home was discussed and felt to be low.

An environmental health inspection was undertaken to ascertain whether appropriate procedures and practices were in place to minimise the potential for food borne contamination or transmission.

Faecal specimens were obtained from 5 crew members following the meetings on 27 July. Specimens were referred for microscopy, culture and viral studies to Royal Darwin Hospital Pathology Service. Specific organisms requested for identification were Salmonella, Campylobacter, Shigella, Giardia, Cryptosporidium, adenovirus, astrovirus, norovirus, enterovirus and rotavirus.

Late in the day of July 27 a meeting was held at CDC, Darwin that included CDC staff (Director of CDC, Head of Surveillance and Enteric Disease Epidemiologist), engineering and construction company management (Quality, Health, Safety and Environment Officer and Industrial Relations Coordinator) and national industrial relations officials. An interim report detailing the outbreak, likely cause, and recommendations for the prevention of further cases on the barge was provided by the CDC outbreak team and discussed.

Results

Information obtained from the meetings on the barge on 27 July enabled the following description of results. Between 4 - 6 July, prior to departing Singapore, the crew members were free to move on and off the vessel and eat on shore. During this time 8 cases presented to the barge clinic with symptoms of diarrhoea and vomiting. There were anecdotal accounts, given to the CDC outbreak team at the meetings on board the barge, of other people boarding with diarrhoeal illness and of less than ideal sanitary conditions in the ablutions area. Specific attention to cleaning of the ablutions area. Specific attention to cleaning of the ablutions was undertaken over the following days at the request of union representatives on board.
Between 7-19 July, the barge was under tow from Singapore to Bali with 181 people on board. The peak of the outbreak occurred in the first 5 days with 65 people presenting to the barge clinic with gastrointestinal symptoms. The barge medical officer described the typical illness profile as abdominal cramps, followed by nausea, then loose explosive bowel actions with or without vomiting, and resolution by 24 hours. Neither myalgias nor fever were a feature. Presumptive antibiotic treatment with doxycycline for a sample of patients made no discernable difference to the course of the illness. The doctor estimated there were 15-20 people with similar symptoms that he spoke to around the barge but they did not attend the clinic. He also described 2 cases with differing clinical profiles. One of these cases disembarked in Bali where he was diagnosed first with amoebic dysentery. On return to Australia he was hospitalised in Perth with salmonellosis. The second case had more severe symptomatology when compared to the other cases, and he responded promptly to intravenous antibiotics and hydration. The overall attack rate, counting cases as clinical presentations to the clinic for the time period 7 – 19 July when the barge was under tow from Singapore to Bali, was 42% (76/181).

On recognising that a greater than usual number of gastrointestinal illness cases had occurred within a short space of time, the barge medical officer undertook measures to investigate the outbreak. These included multiple reviews of the food handling chain from storage, to preparation, serving and consumption. No breaches in standards were identified. Two food handlers developed symptoms during the course of the outbreak and were immediately excluded from their duties. While no systematic food histories were administered, case histories did not suggest any particular food source. On observation, the doctor looked for, but was unable to detect, clustering amongst social groups, sleeping quarters, or port or starboard ablution block use.

Measures instituted by the barge medical officer, occupational health and safety officer and barge captain to contain the outbreak included educating crew at shift handover with verbal and written information about the importance of hand washing. A consistent supply of paper towels in toilets and freshly laundered towels daily for personal use was ensured and extra cleaning of kitchen, mess and ablution areas commenced. The air-conditioning cycle was changed from recirculated to external. As the medical officer could not be certain that the large bottles of water used in multi-use dispensers were not the source, he had these sites closed. As an alternative, smaller personal size bottles of water were freely available for consumption. He sent a sample of these for microbiological examination in Indonesia. The results were negative. Finally, the chlorination levels of the desalinated water not used for consumption were tested and found to be satisfactory.

On 19 July, 30 people disembarked in Bali and the barge continued on to Darwin between 20 – 26 July. While under tow to Darwin another 7 cases reported to the clinic with 3 days between clusters. The attack rate for this period was 5% (7/151).

The environmental health inspection of the barge on the 27 July by the CDC Environmental Health Officer identified only one hand-washing basin in the kitchen area; the condiment refrigerator was not maintaining the appropriate temperature; inadequate storage facilities for cleaning equipment; and a lack of paper towels in the ablutions area. Food handling and storage practices were otherwise adequate. There was no evidence to link the outbreak to food or water. The sanitary and ablation area were clean and well maintained. Appropriate cleansers and sanitisers were in use.

During the peak of the outbreak it was not possible to refer faecal specimens to a pathology laboratory. On arrival in Darwin Harbour 5 specimens were collected; 2 from cases on day 2 of their illness, 2 from cases who had been ill at the peak of the outbreak but were now asymptomatic and 1 from the case who had had a more severe illness and responded to antibiotics. Five days later 2 specimens were reported as PCR positive for norovirus, 1 obtained from a symptomatic crew member and the other from an asymptomatic crew member who had been ill during the peak.
Figure 1. Epidemic Curve of gastrointestinal cases presenting to barge clinic

Discussion

The nature and pattern of symptoms and the temporal relationship of cases closely fulfilled Kaplan’s epidemiological criteria and as such supported a viral cause, such as norovirus, for this outbreak. These criteria are: 1) a mean (or median) illness duration of 12-60 hours; 2) a mean (or median) incubation period of 24-48 hours; 3) more than 50% of people affected and 4) no bacterial agent previously found.

Norovirus, previously known as Norwalk-like virus, may be transmitted by a contaminated food or water source, or from person-to-person by direct faecal-oral, fomite or airborne spread. Close living quarters and periodic change in the susceptible population give rise to ideal environments for the propagation of outbreaks in vessels-at-sea. Norovirus outbreaks on cruise and other ships are well described in the literature.

Information from such reports helped us advise the company and industrial relations personnel even though laboratory confirmation was not received until after the barge inspection.

The initial source of infection was unlikely to be a barge food item as crew were mainly eating on shore in Singapore at that time. A crew member boarding with the illness is more likely to have been the index case, particularly as the epidemic curve (Figure 1) suggested person-to-person transmission. Furthermore contaminated surfaces and subclinical cases were considered responsible for the continued occurrence of cases well after the peak. The potential for continued transmission among new crew boarding in Darwin therefore required attention. Even though more than 100 new crew were due to board in Darwin over the next few days, the investigation team and CDC, Darwin did not recommend quarantining the vessel or original crew. It was considered that the measures already undertaken by the company, as well as implementation of our additional recommendations (see box), would be adequate to prevent further transmission in a situation where the outbreak appeared to be diminishing.

In addition, recommendations to install extra hand washing basins and storage facilities for cleaning equipment in the kitchen were made, as well as advice to properly regulate the temperature in the condiments refrigerator and to ensure adequate supplies of paper towels for hand drying were available.

The outbreak did not continue when the new susceptible crew boarded. It may have been at its end at the time of our investigation as a result of the barge medical officer’s investigation and interventions. Alternatively, the implementation of our additional recommendations may have stopped further transmission.

Conclusion

Although gastrointestinal illness outbreaks on vessels-at-sea are well described we had little prior experience in managing an outbreak of this nature. Thus the experience and knowledge gained during this investigation will help to improve our capacity to manage future outbreaks on vessels-at-sea. By its very nature a barge
Recommendations to prevent and manage gastrointestinal illness outbreaks on board ships and other vessels-at-sea2,8-10

- If someone is ill with gastroenteritis prior to boarding the vessel consideration should be made to postpone boarding until symptoms resolve.
- Ensure hands are washed frequently with soap and water. Rub all surfaces of lathered hands together vigorously for 10 seconds and then thoroughly rinse under a stream of water.
- Adopt a forearm tap as a method of greeting instead of a handshake.
- Have a designated person to serve food to avoid multiple handling of serving utensils.
- Isolate cases from other passengers whilst symptomatic, preferably in a single room equipped with an ensuite.
- People who clean areas contaminated with faeces or vomitus should wear gloves and masks.
- Enhance the sanitation of high usage areas such as ablution blocks, eating areas, recreational areas and external features such as doorknobs and stairwell railings using the following cleaning instructions. This may need to be done prior to new passengers boarding.
- Wash down all potentially contaminated areas with hot water and detergent, rinse with clean water and then sanitise with sodium hypochlorite solution of 1000ppm. Leave sanitiser for 10 minutes and then rinse with cold water.
- Wash, rinse and sanitise all kitchen equipment used by people known or suspected to be infected. Immerse all food contact surfaces into water that is at least 82°C for 2 minutes, or in a solution containing the equivalent of at least 100ppm available chlorine for at least 3 minutes at 50°C. Use a chemical sanitiser at 200ppm available chlorine on any equipment that cannot be immersed in water.
- Use brushes and disposable cleaning fabrics rather than reusable cloths, rags and towels.
- Exclude ill food handlers from work for 48-72 hours after resolution of illness.

such as this is a high risk work environment. Occupational, health and safety officers work diligently at accident reduction and the attending medical practitioners are skilled in the management of both minor and major trauma. It is expected that the knowledge gained by the offshore engineering and construction company following this outbreak will prompt them to continue to give high priority to their occupational health and safety policies and practices. As testified by the barge doctor, knowledge and skills in public health are just as important as the management of trauma in such environments.

Acknowledgements

Dr Peter Markey and Dr Vicki Krause

References

2. Centers for Disease Control and Prevention, "Norwalk-like viruses:" public health consequences and outbreak management. MMWR, 2001. 50(9)
The utility of screening Royal Darwin Hospital health care workers for tuberculosis infection with a two-step Mantoux test.

Nathan Zweck & Belinda Farmer, CDC, TB Unit, Darwin

Introduction

This article describes the rationale for the recommendation that health care workers (HCW) in the Northern Territory (NT) be screened with a two-step, rather than a one-step Mantoux test. Adherence to, and results of, the screening since January 2003 are analysed in order to assess the utility of two-step testing in this population, and to judge whether the practice should continue. Recommendations are offered for discussion by NT Centre for Disease Control (CDC) staff in the lead-up to the CDC conference in October 2004.

Background

Method and rationale for screening HCW

Screening for tuberculosis (TB) with a single baseline Mantoux test has been a condition of employment, promotion and transfer for HCW in the NT Public Service since 1991.1 Where a Mantoux test is positive the HCW is recommended to present to the TB Unit for a medical officer review and a chest x-ray, but these steps are not mandated by the Public Service Act. If active TB disease is ruled out, a diagnosis of latent TB infection (LTBI) is made. LTBI is a state of harbouring dormant or ‘latent’ TB bacilli in the body, and depending on individual characteristics (age, chest x-ray fibrosis, time since infection occurred, co-morbidities, immunsuppressing medications), confers varying risks for the future development of TB disease.

The rationale for TB screening of HCW and other staff who work in clinical settings has both public health and individual dimensions. Reasons include:

1. for staff who are initially not infected (Mantoux negative) annual monitoring is done to detect new infection (Mantoux conversion) and to prevent early progression to disease with preventive treatment;
2. to diagnose pre-existing latent TB infection (LTBI) to provide an opportunity for discussion of the individual’s risks of future TB, and ways of managing this risk; and
3. to detect active TB disease in new staff at the commencement of employment and offer curative treatment, to protect patients and fellow workers from being infected by them.

The two-step Mantoux test

The Mantoux test as a diagnostic tool has remained essentially unchanged since its introduction over a century ago. A positive test suggests infection with Mycobacterium tuberculosis, but cannot in isolation discriminate between whether the infection has progressed to active disease or indicates latent infection with no active disease (LTBI).

As there is no gold standard test for LTBI the sensitivity of the Mantoux test for LTBI cannot be calculated. Since the Mantoux relies on an intact delayed type hypersensitivity (DTH) response, suppression of cell mediated immunity can produce false-negative results. A single Mantoux test given many years after an individual has acquired TB infection may be falsely negative due to waning of the DTH response over time. In diagnosis of active TB it lacks sensitivity as various studies report up to 32% of HIV-negative persons with culture-positive proven TB disease have negative results.2

One way to improve the sensitivity of the Mantoux test (achieve more true positive results), and to establish those who are truly infected with TB, is to repeat the test 1 to 3 weeks later after an initial negative test. This is referred to as the two-step Mantoux test. The first test, although negative, awakens the waned immune response from a past infection, and a boosted, positive result may occur on the second Mantoux.

Those who work or reside in environments where TB transmission is likely to occur should undergo annual Mantoux testing if their initial test is negative, in order to detect Mantoux conversion (new infection) as early as possible.3
The advantage of the two-step Mantoux test at baseline screening is to avoid misinterpreting a positive test 1 year later as a conversion due to newly acquired infection when it may simply be a boosted response to an old infection (where risk of progressing to TB disease is less).4

The downside of the two-step Mantoux test is that 2 more visits are required which is inconvenient to the person being tested and increases the workload sustained by TB Unit staff.

Methods

New employees of the Royal Darwin Hospital (RDH) receive the RDH Staff Health Assessment Screening Form as part of their employment orientation package. This requires signed documentation of a previous positive Mantoux result, or of a current Mantoux result by staff of the TB Unit, and is then returned to the staff member. A questionnaire regarding immunisation status is included on this form for discussion with the staff immunisation clinic.

Staff who attended had a Mantoux test performed if there was no contraindication (previous positive Mantoux or previous TB) according to the method described in the Guidelines for the Control of Tuberculosis in the Northern Territory, December 2002,5 and the result was read 3 days later. The cut-point for a positive result was 10mm. Those with a negative result were counseled and asked to return 2 weeks later for a repeat test if there was no history of a negative test in the preceding 12 months.

The TB Unit in Darwin has a list of 61 referral-in category codes, and this is recorded on a daily worksheet each time a Mantoux is given, along with the client’s name and HRN. Mantoux data was manually extracted from this worksheet by one author (BF) for persons with codes corresponding to HCW employed in the RDH who presented for a Mantoux test in 2003.

Results

In 2003, 236 HCW were given an initial Mantoux test (Mantoux 1, Figure 1). Of these, 24 (10%) failed to return to have the result measured, and of 212 who did return, 55 (26%) had a positive result and 157 (74%) had a negative result.

Those with negative results were advised to return for a second Mantoux test (Mantoux 2) and 61 (39%) complied with this recommendation. Of 61 who underwent the two-step test, 54 (89%) remained negative, (thus requiring annual Mantoux testing) while 7 (11%) boosted their initial negative to a positive result. The mean difference between the first and second Mantoux results for the 7 boosted tests was 10mm (standard deviation 2.08mm, range 7-13mm).

Figure 1. Flow chart showing adherence to, and results of, the stages of two-step Mantoux screening among health care workers in the NT in 2003.

Discussion

Utility of the two-step Mantoux test

There is a relatively high prevalence of boosting (7/61, 11%) among initially Mantoux negative HCW who underwent a second test in this cohort. This compares with 6% and 2.5% respectively (Table 1) among staff of medical
centres in California\(^6\) and Michigan\(^7\) in the United States of America (USA). Boosting is known to be associated with prior BCG vaccination, older age, and overseas birth,\(^8\) and differences in these factors may account for our findings, particularly the fact that the USA, unlike Australia, does not have a BCG vaccination program.

Notable also is the higher overall prevalence of baseline Mantoux positivity in Darwin HCW (29%) compared with the US centres (7% and 6%). However, it is the observation of the TB clinic staff that HCW commencing employment in the RDH have often had clinical experience in remote Australian settings with high burdens of TB, or were born or have worked overseas in countries with high TB rates.

The fact that 5 units of tuberculin are used in the Mantoux test in the USA compared with 10 units in Australia is unlikely to be clinically important.\(^9\)

As a result of the two-step Mantoux recommendation, 7 individuals in Darwin with boosted results have appropriately received clinical and radiological evaluation for active TB at the start of their employment, rather than one year or more later on serial testing. Accurate counseling about risks of their LTBI status has been possible immediately, and rational management decisions made.

Better compliance with the two step test would be of benefit to more health care workers in establishing LTBI in a timely fashion and eliminating the need for serial Mantoux in those who are true positives. For example, if the 157 who had been advised to undergo a two-step Mantoux had done so, (and assuming the same prevalence of boosting, i.e. 11%), the number of positive tests due to the second step would have increased from 7 to 18. This implies that up to 11 individuals in the pool of 96 who underwent only one-step, could boost to a positive result one year later after serial testing. Unfortunately, they would be advised that a recently acquired infection could not be excluded, and that completion of 9 months of isoniazid is very strongly recommended. Apart from the personal costs of an inaccurate baseline Mantoux, there would also be a loss of public health intelligence which results from confidently detecting new infection in HCW. This surveillance can point to locations in the hospital where transmission has occurred, and prompt action to strengthen engineering or administrative measures to increase protection of staff and patients.

The benefits of the two-step test extend also to the majority who underwent a second test (89%) and whose results were negative a second time. They are more assured of not being infected at the commencement of employment, have a heightened awareness of the need for serial testing, and if conversion occurs, will be in no doubt that the benefits of preventive treatment outweigh the risks.

Another index of the utility of the two-step method is the proportion of all positive tests (after Mantoux 1 and Mantoux 2) which were detected by the second test. This is highly dependent on the level of compliance with the second step, since low adherence will result in fewer positive tests from Mantoux 2 relative to Mantoux 1. Therefore, it also reflects acceptance

<table>
<thead>
<tr>
<th>Population</th>
<th>Proportion of all tested and read who were PPD positive</th>
<th>Prevalence of the booster effect in those having a second test</th>
<th>Proportion of all positive results detected by the second test</th>
<th>Conclusion about 2-step baseline Mantoux for HCW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Royal Darwin Hospital, NT, Australia</td>
<td>62/212 (29%)</td>
<td>7/61 (11%)</td>
<td>7/62 (11%)</td>
<td>Targeted recommendation</td>
</tr>
<tr>
<td>Veteran’s Affairs Medical Centre, California, USA(^7)</td>
<td>45/619 (7%)</td>
<td>6/97 (6%)</td>
<td>6/45 (13%)</td>
<td>Recommended</td>
</tr>
<tr>
<td>Inner city medical centre, Michigan, USA(^8)</td>
<td>243/3896 (6%)</td>
<td>6/241 (2.5%)</td>
<td>6/243 (2.5%)</td>
<td>Not recommended</td>
</tr>
</tbody>
</table>
by HCW of the second step. In Darwin 11% (7/62) of all positive results were detected by the second Mantoux, but if all 157 indicated to have a second Mantoux complied, and the prevalence of boosting was the same, 25% (18/73) of all positive tests would be attributed to Mantoux 2.

**Compliance of HCW with screening requirements of the Public Service Act and recommendations**

- **Administration of a baseline Mantoux test**

Although a denominator for all types of HCW newly employed in the RDH in 2003 is not available, it is known that 434 nurses and doctors were newly employed by the NT government. Some of these would not have been tested due to documentation of a previous positive result, while others may have had very short appointments of a few weeks and departed the NT before screening was possible. Since only 236 HCW had a first Mantoux given, it is likely that a substantial proportion ignored the requirement to attend. Unpublished data for the first 6 months of 2003 showed that of 173 nurses newly hired in that period, only 58 (33%) attended for an initial Mantoux test.

- **Reading the result of a Mantoux test**

Since 10% of those who had an initial test did not return to have it read, it is possible that HCW have self-assessed the absence of a visible reaction as evidence of a non-reactive result. However, measurement of results is not visual, but relies on the ball-pen technique which is not possible to self-administer with accuracy. More intensive counselling at the time of test administration about measurement techniques, and the need for follow up of negative tests might result in more HCW returning for results.

In 1991 the NT Public Service Commissioner wrote a letter to the Secretary of DHCS outlining administrative procedures which should accompany Determination 36 of 1991 relating to baseline Mantoux testing. It is clear from analysis of this letter that the original intention was for screening to occur and be completed **prior** to commencement of employment, and that confirmation of recruitment depended on this. These procedures have waned and need to be revisited if it is acknowledged that Mantoux screening is appropriate.

- **Compliance with the two-step recommendation**

The two-step process for baseline Mantoux testing is recommended by local TB Guidelines but is not mandated by the Act. The uptake of Mantoux 2 by 39% of eligible HCW is a credit to the counselling skills of Darwin TB Unit staff, and the informed decision making of the HCW concerned. It compares favourably with uptake proportions of 17% in California and 7% in Michigan.

- **Clinical review, chest x-ray, and annual Mantoux testing**

Baseline Mantoux testing is compulsory under the dictates of the NT Public Service Act, but completion of a chest x-ray and clinical review for reactors, and annual testing for non-reactors is not. This study did not aim to evaluate adherence to these strategies, but it is logical that mandatory and accurate baseline Mantoux testing only makes sense if the measures it indicates are implemented. If they are not, then screening is a waste of resources. Our impression is that the majority of Mantoux reactors attend for evaluation, but very few Mantoux non-reactors return annually for serial testing.

Victorian TB guidelines state that institutions which admit one or more patients with infectious pulmonary TB per year should assess the TB risk to groups of HCW by prospectively measuring Mantoux test conversion rates. NT hospital TB admission rates qualify for this recommendation with an average of 12 cases hospitalised per year over the past 16 years. Surveillance among hospital HCW tested 6 monthly following a negative baseline two-step Mantoux test showed that conversion occurred in 1.2% in Atlanta (rate 0.38 per 100 person years), and 1.5% in New York and Boston (1.6 per 100 person years). In Atlanta the annual conversion rate for nurses prior to the implementation of a TB infection-control program was 13%. The infection control measures in these hospitals were similar to those employed at the RDH – a respiratory isolation policy, negative pressure respiratory isolation rooms, and personal respiratory protection equipment.
If, as is likely, similarly low rates of transmission and conversion are occurring among RDH HCW, serial testing, and therefore two-step baseline testing could prudently be restricted to HCW working in areas where the risks are highest. It is well documented that multifaceted infection control measures including negative pressure isolation rooms can rapidly and significantly reduce conversion rates. However, Menzies and others have demonstrated that the most important determinant of HCW conversions was the poor ventilation (less than 2 air exchanges per hour) in non-isolation general patient rooms (in contrast to designated isolation suites) reflecting exposure to undiagnosed TB patients. They conclude that “early detection is the most important intervention to reduce nosocomial transmission”, and this relates to the clinical index of suspicion of TB, prompt isolation when TB is in the differential diagnosis for a respiratory illness, and timely mycobacteriology reports. Therefore, HCW who are to be deployed in regional hospitals in the NT which currently do not have negative pressure isolation rooms could still be screened in a targeted way, so long as other TB infection control strategies are in place.

Although of 599 cases of TB notified in the NT from January 1989 until March 2004 only one has been diagnosed as a direct result of pre-employment screening, 9 additional cases diagnosed by other means have had employment in the health care sector as a risk factor (source - NT enhanced TB notification database). However, prior to compulsory HCW screening in 1991, infectious pulmonary TB was detected in a HCW in the NT who was working with at risk patients. Such a scenario, although rare, is potentially catastrophic for vulnerable patients, other HCW, and the reputation of the institution. While baseline Mantoux screening is not a diagnostic test for TB disease, if compliance with attendance can be improved, value could be added to this encounter by an enquiry about the presence of a cough with a duration exceeding 3 weeks. Symptomatic individuals could be fast-tracked to a clinical review, chest x-ray, and sputum mycobacteriology, regardless of the eventual Mantoux result.

Conclusions

To formulate recommendations for future practice, the following must be considered,

- two-step Mantoux testing does give a superior baseline result for HCW who are to be serially tested, but its universal practice is logical only if serial testing comprehensively occurs;
- the specific context of the RDH as a facility which does diagnose and accommodate several infectious patients per year, but also one with best practice isolation policies and engineering controls which probably result in a low risk of TB transmission;
- the opportunity cost of strengthening aspects of HCW screening when weighed against alternative strategies for reducing TB incidence in the NT which may have a greater impact;
- different groups of HCW have heterogeneous levels of risk, and a blanket policy for all may not be the best use of resources.

Recommendations

1. Leave the current legislation unchanged (one baseline Mantoux required), but implement the administrative procedures where attendance for TB screening is reported to the recruitment office to ensure compliance -
- allow 4 weeks after commencement of duties for completion of Mantoux screening rather than the pre-employment requirement;
- re-design the current RDH, and other NT hospitals, Staff Health Assessment Screening Form to include an excerpt of the Act, and a return compliance slip for endorsement by TB Unit staff and forwarding to the recruitment office;
- implement an auditing system which flags the lack of endorsement for an individual 4 weeks after commencement of their employment, and notifies the Public Service Commissioner accordingly.

2. Create 9 new referral-in codes applying to the small sub-sets of HCW at the highest presumed risk of acquiring TB infection –
- staff in the mycobacteriology laboratory, mortuary, intensive care unit, emergency department, bronchoscopy suite, TB Unit, respiratory function studies unit, staff of Clinic 34 caring for HIV positive clients, and physiotherapists performing induced sputum procedures;
♦ restrict, and intensify, educational efforts to obtain a two-step baseline in these groups only;
♦ create a recall, reminder, and incentive system for these codes to ensure annual serial testing of baseline non-reactors occurs;
♦ produce an annual de-identified report of the Mantoux conversion rates to inform preventive measures;
♦ widen the two-step recommendation to other HCW groups if transmission is consistently documented in the high-risk groups.

3. Introduce simple symptom screening (cough >3 weeks duration) for active pulmonary TB at the time of administration of a baseline Mantoux test.

References
1. Public Service Act, Determination No. 36 of 1991
3. Guidelines for preventing the transmission of mycobacterium tuberculosis in health care facilities, 1994 MMWR October 28, 1994
5. Guidelines for the Control of Tuberculosis in the Northern Territory, Dec 2002 Northern Territory Government and Community Services.
6. Sherman RA and Shimoda KJ Tuberculosis tracking: Determining the frequency of the booster effect in patients and staff, American Journal of Infection Control 2001;29 (1); 7-12
8. Menzies D. Interpretation of Repeated Tuberculin Tests Boosting, Conversion, and Reversion AM J Respir Crit Care Med 1999;159:15-21

The Territory two step – enhancing detection of latent Mycobacterium tuberculosis infection in HIV clients.

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The Northern Territory (NT) has the highest rate of TB of any Australian jurisdiction with the burden of disease predominantly in the Indigenous and overseas born populations. High proportions of these groups also have latent TB infection (LTBI), and coinfection with HIV is the greatest known risk factor for reactivation to TB disease. Previously the NT AIDS and STI Program has screened HIV seropositive clients who are newly diagnosed or newly arrived in the NT for TB. This screening varied depending on the preference of the incumbent physician.
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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*CDC and *Menzies School and RDH DHCS Darwin NT

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 x109/L with a mild lymphopaenia of 0.9, haemoglobin of 122g/L and platelets of 170x109/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 \textit{Ae. aegypti}, and it seems certain that this species has been responsible for many of the Asian urban epidemics.

All \textit{Culex} species so far tested including \textit{Cx. quinquefasciatus} have been found refractory to infection with chikungunya virus.

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

Some populations of \textit{Ae. albopictus} have been shown to be better vectors than populations of \textit{Ae. aegypti}, while other populations appear to be less efficient. Hence, \textit{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 \textit{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 \textit{Ae. aegypti} should be eliminated by reducing the number of water containers, and control of adults and larvae may be necessary. Surveillance of \textit{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.\textsuperscript{3} The virus is considered to be a relatively recent introduction into South East Asia and an increasing public health problem.\textsuperscript{3}

In Indonesia, chikungunya, first emerged in Bandang, West Java, in December 2002,\textsuperscript{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.\textsuperscript{3}

As there is currently no \textit{Ae. aegypti} in the NT except in Tennant Creek, and no \textit{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 \textit{Aedes} mosquito vectors such as \textit{Ae. aegypti} and \textit{Ae. albopictus}.

References


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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 a 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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Should Acute Rheumatic Fever and Rheumatic Heart Disease be nationally notifiable?

Philippa Binns and Vicki Krause, CDC, Darwin

Introduction

Acute Rheumatic Fever (ARF) following Group A streptococcal infection of the throat, and possibly skin, is a serious disease, especially in children. It can have significant long-term consequences including death due to the heart valve damage of rheumatic heart disease (RHD). ARF is rare in developed countries, yet Indigenous Australians have one of the highest recorded incidence rates in the world, with children being disproportionately affected.

ARF and RHD are notifiable conditions in the Northern Territory (NT) and Queensland, and information is collected in Western Australia. The NT incorporates the data into a register as part of a program to aid service delivery and improve outcomes for people with these conditions as well as to establish the burden of disease. However, collective or comparative analysis is not possible between jurisdictions as data is not standardised nor collected in a co-ordinated fashion. Also, those living in other jurisdictions are not identified and included.

National notification would be the most appropriate means of surveillance because it would identify those areas in Australia deserving attention and resource allocation to address these problems.

Public Health Principles

For a disease to be considered nationally notifiable in Australia, it must follow these broad public health principles:

1. It must be identifiable (i.e., it must be a recognisable disease).
2. It must be preventable.
3. There must be the potential for action, either through prevention or treatment (via regulation, policy and/or education).
4. There must be identified population or sub-population targets, at a national, regional or global level.
5. Data must be usable (i.e., the information gathered should serve a useful purpose at a local and global level).

These principles are discussed below with respect to ARF and RHD:

1. Identifiable

ARF is diagnosed by the modified Jones criteria, which include clinical signs and laboratory results. In the NT, these criteria have been incorporated into the Rheumatic Fever case definition under the NT Notifiable Diseases Act:  

Case definition for a First Episode Acute Rheumatic Fever in the NT

Two major manifestations or one major and two minor manifestations
AND
Evidence of recent streptococcal infection (positive Group A streptococcal throat culture, elevated ASO titre or AntiDNAase B)

Case definition for a Recurrence of Acute Rheumatic Fever in the NT

People with past rheumatic fever or established rheumatic heart disease require only one minor manifestation
AND
Evidence of recent streptococcal infection (provided other diagnoses have been excluded)

Where Major Manifestations are
Carditis, polyarthritis, chorea, erythema marginatum, subcutaneous nodules
And Minor Manifestations are
Arthralgia, fever, raised ESR, raised C-reactive protein, prolonged PR interval on ECG
The Northern Territory Disease Control Bulletin Vol 11, No. 3, September 2004

The decline in ARF in developed countries during the 20th century has led to a lack of awareness in identification of this diseases by medical professionals in mainstream practice. Given the high rates in the Indigenous population, all practitioners Australia-wide need to maintain an index of suspicion for overt, mild or subclinical cases of ARF and RHD so that cases continue to be identified for earlier intervention.

RHD can be identified by characteristic features on echocardiography.

2. Preventable

Primary prevention of ARF is possible by appropriate antibiotic treatment of Group A streptococcal infections. Once diagnosed, secondary prevention with penicillin prophylaxis is essential and effective in preventing disease recurrences and progression to RHD. The decline of ARF in affluent populations has been attributed largely to economic development and improved living conditions, with perhaps a small contribution from antibiotics and altered virulence of the Group A streptococcal strains. There is no evidence for a predisposition based on ethnicity. Improvement in economic status and living conditions for those at risk today would contribute to the decline as witnessed in the time of post World War 1 prosperity in temperate Australia.

3. Potential for action

ARF and RHD programs incorporating registers and education are recommended by WHO and have been underway in the NT since 1998. A business plan is under development for a National Rheumatic Fever/Rheumatic Heart Disease register-based control program to improve education, and primary and secondary prevention nation-wide.

Attention to effective policy and programs to reduce the disadvantage experienced by Australia’s Indigenous population with respect to poverty and overcrowding would also contribute to primary prevention.

4. Identified population or sub-population targets

RHD was identified in 1998 as one of the “National performance indicators for Aboriginal and Torres Strait Islander Health”. A target was established to reduce mortality due to RHD by 50% by 2008. Whether remote, rural or urban, all Aboriginal and Torres Strait Islanders are included in these performance indicators and targets.

5. Useable data

ARF and RHD surveillance data would help us understand the distribution of and to identify trends in the diseases nationally. These are specified roles of communicable disease surveillance at a national level in Australia. In turn this would help fulfill another role of national surveillance, which is to guide policy development and resource allocation. Surveillance data would also contribute to a national register-based control program.

Criteria for national notification

When a disease fulfills the public health principles it may be suitable for national notification. The Communicable Disease Network Australia (CDNA) has set out criteria to guide whether a disease should be nationally notifiable.

They are:

1. Feasibility of collection
2. Priority (State/Territory vs. National policies/interests)
3. Immediacy of the intervention that is possible and/or required
4. Outbreak potential
5. Potential for new programs or for refinement of existing programs
6. Maintenance and evaluation of existing and future programs
7. Community/political concerns
8. International concern
9. Maintenance and evaluation of existing and future programs

10. Importance to Indigenous Health

Interestingly, communicability of a disease is not a criterion. However RHD, like haemolytic uremic syndrome (HUS) which is nationally notifiable, is the sequela of a communicable disease. It is therefore appropriate to consider RHD for national notification. The criteria for national notification are discussed below with respect to ARF and RHD.

1. Feasibility of collection

A national case definition for ARF is possible based on the Jones diagnostic criteria. Data are currently collected in the NT and Queensland indicating feasibility in Australia. Looking further afield, ARF was recently recommended to be retained on the schedule of notifiable diseases in New Zealand indicating feasibility and importance at the national level in that country.

The feasibility of data collection for RHD has been demonstrated by the NT ARF/RHD Register. Just as laboratory notifications function throughout the country, echocardiographic notifications could be considered. Cardiologists and radiologists could have flagging built into their systems to recognise patients with RHD and be responsible for notification.

2. Priority (State/Territory vs. National policies/interests)

Cardiovascular health is included in the National Health Priority Areas initiative whose advisory group is the National Heart, Stroke and Vascular Health Strategies Group. One of their priorities for national action is to identify, manage and treat people with RHD. If ARF and RHD are notifiable and appropriate public health action is implemented then identification and management will be facilitated and improved.

3. Immediacy of the intervention that is possible and/or required

Timely education and prophylaxis is essential and recommended to prevent recurrences of ARF and development of RHD.

4. Outbreak potential

Outbreaks of ARF have been well documented in developed countries (e.g. U.S.A., Canada, Italy) with a corresponding higher prevalence of RHD resulting. The Australian Institute of Health and Welfare (AIHW) which is informed by the NT RHD Register reports that in 2002 there were 305 cases of ARF for every 100,000 Indigenous children aged 5–14. Current data documents an endemic problem in the NT; however epidemic potential may still exist in the Indigenous and non-Indigenous populations.

5. Potential for new programs or for refinement of existing programs

The Register program in NT has been established to improve patient care, particularly secondary prevention, by establishing a reminder system for monthly penicillin injections and other clinical follow-up by the primary care system.

A business plan is being developed to improve and expand this to be used as a national program. National notification would help with planning and refining such a program.

6. Maintenance and evaluation of existing and future programs

Notification of ARF and RHD would contribute to understanding the changing epidemiology of the ARF and RHD and therefore contribute to the quality assurance and evaluation of register-based control programs. If a State or Territory did not have a management program, a surveillance program would be maintained by the contribution of epidemiological knowledge gained from notifications.

7. Potential for a high-case fatality rate

ARF and RHD accounted for 242 deaths in Australia in 2001. Indigenous Australians are far more likely to die from ARF and RHD than other Australians. In the last 10 years, of the 63 deaths known to be due to RHD of people on the NT RHD Register, 26 were in those less than 35 years of age.

Notification of ARF and RHD would allow informed, appropriate and targeted public health action, and contribute to the prevention of RHD and associated mortality.
8. Community/political concerns

ARF and RHD affect Indigenous children, a marginalised group in this country. The community and politicians will not be concerned unless those who have knowledge of the problem with a voice and influence in the national forum bring it to their attention. By being nationally notifiable an opportunity is provided to document these preventable, life-threatening conditions and inform the community and politicians, accurately and appropriately.

9. International concern

The WHO Global Programme for Prevention and Control of Rheumatic Fever and Rheumatic Heart Disease and the WHO Global Programme on Cardiovascular Disease recommend developing feasible surveillance methods to assess the pattern and trends of major cardiovascular disease and risk factors, and to monitor prevention and control initiatives.23 A WHO-commissioned review reports the incidence of ARF in Indigenous Australians, and people of the Pacific and New Zealand, is the highest recorded in the world. The prevalence of RHD is second only to sub-Saharan Africa.24 As a nation that has been identified with such statistics by a WHO-commissioned document, the notification of these diseases in Australia would address this international concern.

10. Importance to Indigenous Health

ARF and RHD are almost entirely a health problem of Indigenous people in Australia where preventable deaths are occurring in a young population.

Conclusion

ARF is a consequence of a communicable disease. As discussed above it fulfils all the public health principles for surveillance and criteria for a disease to be nationally notifiable. Accurate surveillance would lead and inform effective public health action, such as a national ARF/RHD register-based program.

Australia’s Indigenous population has the highest incidence rates in the world of ARF yet it is not monitored nationally. This is in stark contrast to our neighbours in New Zealand who acknowledge that their Maori and Pacific Island children are affected disproportionately compared with most developed countries. As a result, ARF has been recommended for retention on their schedule of notifiable diseases.14

Notification and surveillance of ARF and RHD at a national level would contribute positively and effectively to public health action by addressing national priorities and targets aimed at improving the health of a disadvantaged population group living in all areas of Australia; urban, rural and remote.

References


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Amendment to the current Guidelines for the early clinical and public health management of meningococcal disease in Australia.

The Communicable Diseases Network Australia (CDNA) has published an amendment to the guidelines with regard to saliva as a means of transmission of Meningococcal disease.

Available evidence does not support saliva or salivary contact as being an important means of transmission of meningococcal infection.

The following contacts are not recommended to be offered chemoprophylaxis unless they are house-hold, child care or very close contacts (e.g. sexual contacts)1:

- Kissing contacts, even if mouth kissing was involved;
- Food, drink (including drink bottle) sharing contacts;
- Communion cup, lip balm, wind instrument, referee’s whistle sharing contacts; or
- Any other similar low level salivary contacts

1 The amendment in full can be found on the CDNA website at URL: http://www.cda.gov.au/pubs/other/pdf/mening_amend.pdf
Injury Prevention a National Plan for consultation and moving forward in the NT

Steven Skov, Public Health Physician, CDC, Darwin

The National Public Health Partnership (NPHP) has a sub-committee known as the Strategic Injury Prevention Partnership (SIPP). The SIPP provides a forum for leadership in injury prevention in Australia and promotes a consistent, integrated approach to injury prevention, including monitoring and evaluation across all areas of government. The SIPP was responsible for implementing the National Injury Prevention Plan: Priorities for 2001-2003 and the development of a subsequent national plan.


The priority areas in the 2004 draft national plan are:

1. Children (0 – 14 yrs)
2. Emerging Adults (15 – 24 yrs)
3. Older People (65 yrs+)
4. Aboriginal and Torres Strait Islander Communities
5. Rural and Remote Populations
6. Alcohol and Injury

The aims of the consultation are to:

1. Obtain feedback about the proposed direction and content of the Draft Plan and Strategy, including the feasibility of, and priorities for, implementation;
2. Inform stakeholders, communities and interested individuals about the development of the Draft Plan and Strategy; and

Initial consultation is currently taking place across the country. State and Territory based face to face workshops have been conducted and written submissions are being sought with and from a wide range of both government and non-government agencies. National workshops with representatives from all states will be held in October and November with the aim of producing final drafts for fine tuning by the SIPP. The documents will then be passed up through the NPHP to the Australian Health Ministers Council for final approval, hopefully, in April 2005.

As community physician, I am the NT representative on the SIPP and have responsibility to coordinate the consultation within the NT. We have circulated the documents very widely, conducted workshops in Alice Springs and Darwin and will be sending Top End and central Australian representatives to the national workshops.

During the NT workshops ways were discussed to progress the issue of injury prevention in the NT. There was a strong recognition of the need for a more integrated, coordinated and intersectoral approach to injury. Workshop participants felt that the NT was in need of a whole of government approach with a dedicated injury prevention unit working to a high level multi-agency governance committee. In the near future, I intend to consult more widely regarding support for such a strategy, and if possible to play some role in coordinating action towards it.

I invite anyone with an interest in injury prevention, either concerning the national plans or particularly in relation to the Territory, to contact me.

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Update on recommendations for treatment of *Neisseria gonorrhoeae* infection in the Darwin region

*Steven Skov, Public Health Physician, CDC, Darwin*

The last edition of the *Bulletin* reported on a change to the recommended treatment for gonococcal infection in the Darwin urban and rural regions from oral amoxycillin and probenecid to injected ceftriaxone. This was necessary because of the discovery of a number of cases of local transmission of penicillinase producing *Neisseria gonorrhoeae* (PPNG) in Darwin. All health care providers were notified of the change and the reasons for it, and were encouraged to take culture specimens whenever possible when seeing patients who may have gonorrhoea. This recommendation was to be reviewed after 3 months.

Since the initial outbreak, there have been no further cases of local transmission of resistant gonorrhoea observed. There have been some cases in people who acquired the infection overseas. However, it was not possible to find all the sexual partners of the original cohort of people who had resistant infection. It is felt that not enough time has yet elapsed to be confident that any local transmission of resistant gonorrhoea has ceased.

Therefore, the AIDS/STD program recommends (see box) that all people in the Darwin region who may have gonorrhoea continue to be treated with 250mg of IMI Ceftriaxone. This includes immediate syndromic treatment of those people with symptoms and signs consistent with gonorrhoea (as well as azithromycin to cover chlamydial infection) and people with confirmed gonorrhoea (unless a culture result clearly indicates penicillin sensitivity). In all other regions of the NT, people with possible gonorrhoea should continue to be treated with amoxycillin and probenecid as per the long standing protocols. Ceftriaxone should be used for all patients throughout the NT who have a history of sexual contact outside the NT.

If there is a contraindication to ceftriaxone the possible alternatives are single doses of 500mg oral ciprofloxacin or 2g of oral azithromycin. However, neither of these should be used as a routine and patients should be seen within a week to ensure they are cured. There is considerable ciprofloxacin resistance in SE Asia and the rest of Australia already and azithromycin at this dose will cause significant gastro-intestinal side effects in up to 30% of patients. In both cases every effort should be made to obtain a culture specimen prior to treatment.

The AIDS/STD program will continue to monitor the situation and will review this recommendation at the end of the year. All queries, requests for information or suggestions, should be directed to either the AIDS/STD program in CDC on 89228606 or Clinic 34 on 89992680.

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**Treatment recommendations for all people in Darwin region with possible or confirmed gonorrhoea.**

Person with symptoms or signs of possible gonorrhoea: immediate treatment with 250 mg of IMI Ceftriaxone and 1 gm of oral Azithromycin (to cover chlamydial infection).

Person with laboratory confirmed gonorrhoea: 250 mg of IMI Ceftriaxone (unless culture result indicates organism sensitive to penicillin).

* This recommendation will be reviewed at the end of 2004.
Darwin’s Meningococcal C School Based Vaccination Program 2004

Rosemary Day & Amy Ryan, Immunisation Program, CDC, Darwin

The Meningococcal School Based Vaccination Program has been mapped out encompassing Darwin, Palmerston and ‘Outer Darwin’ with the inclusion of Dundee Beach Primary and Taminmin High Schools.

We, the Meningococcal C Team (registered nurses (RNs) Rosemary and Amy), head off daily to a pre-scheduled school with our equipment and vaccines from Royal Darwin Hospital (RDH) pharmacy, whose staff has been extremely supportive towards the program. Kylie Ryan, the administrative officer for the program, contacts the schools, organises delivery and return of consents, checks for previously vaccinated students on the Meningococcal C and Community Care Information System databases, highlights and removes any “NO” consents as well as any who have been previously vaccinated for meningococcal C. This aims to avoid any inadvertent un-consented or unnecessary vaccination.

Once this is completed, consents are checked to ensure legality. Parents are phoned when necessary to clarify questions regarding the consent. There has been some confusion regarding previous vaccinations. To clarify this, if the child has received any of the following conjugate meningococcal vaccines (MenCCV): Meningitec, Menjugate or NeisVacC-C (used in the school based Meningococcal C Program), they have been vaccinated against meningococcal C with a long acting vaccine and do not need further vaccination. The meningococcal polysaccharide vaccines (4vMenPV), Mencevax or Menomune, provide vaccination for serogroups A, C, W135 and Y, but are shorter acting vaccines requiring repeat vaccination in 3-5 years. 1 Therefore, vaccination with MenCCV should be given to ensure long term immunity. A spacing of 6 months after the (4vMenPV) is required before administering the MenCCV.

Each school is contacted by telephone (or occasionally in person) prior to the vaccination date to ascertain the number of late returns of consent forms (the lovely, never to be forgotten bright lime green ones), in order to confirm that an adequate quantity of vaccine has been ordered and to ensure adequate facilities are available.

Arrival at each school engenders nervousness, sweaty palms and anxious faces in students and the occasional teacher. The actual vaccination takes minimal time with a 15-minute observation period. With good traffic control we can vaccinate up to 150 children before lunch. In the 3-month period June-August 2004, we vaccinated 3,417 students from 22 schools. We have had very few adverse events following immunisation, those reported being a single episode of prolonged muscle flaccidness (restricting use) for 1-2 days post vaccination and one child with heat, redness, pain and slight swelling at the injection site one day post vaccination. In addition, one child fainted, followed by seizure like behaviour, was sent to RDH via ambulance for further medical assessment/treatment and returned with his mother soon after to reassure us he had recovered. All above cases made speedy, full recovery with no further action required.

The target range for eligible recipients of the Meningococcal C Program is the 7-15 year olds, which unfortunately has proven restrictive by excluding transition year of 5-6 year olds who may not have received the vaccine last year, therefore parents of this age group are recommended to take their child to the Community Care Centres for vaccination.

Some of the challenges faced by the RNs implementing the program are playground rumours of the actual vaccination process, which adds to student anxiety. However once completed we often hear ‘is that all it is’? and a sigh of relief expressed by the student, and much appreciated are the odd thank you and smiles we receive from the students.

It is anticipated that all eligible students with consent will be vaccinated by 10 December 2004.

Reference

Environmental Health Program 2003/2004

Xavier Schobben, Environmental Health Program, CDC, Darwin

The Environmental Health Program prepared this report for the Department of Health and Community Services (DHCS) annual report. Due to the allocation of space for the Centre for Disease Control and Environmental Health being condensed by approximately 50% we were unable to present this information in full so have taken this opportunity to document the work of the program for the last 12 months.

Overview

Environmental Health is encompassed within the broader area of public health. The Environmental Health Program is involved in the assessment, correction, control, and prevention of environmental factors adversely affecting human health. Environmental health practice provides opportunities for improved health outcomes and working towards health promoting environments.

Objectives of Environmental Health are to:

- develop, implement and enforce environmental health, food safety, radiation protection and poisons control legislation.
- monitor, audit, inspect and risk assess all commercial premises including take away food shops, markets, restaurants, boarding houses, beauty salons, hairdressers, and ear and body piercing establishments.
- work with other DHCS programs and with other agencies, communities and key stakeholders to improve environmental health outcomes and support ‘at risk’ groups.
- action and resolve complaints received regarding environmental health, radiation safety, poisons control and food safety matters.
- assist in the development of large scale housing and environmental health infrastructure projects under both the National Aboriginal Health Strategy – Environmental Health Program (NAHS-EHP) and the Indigenous Housing Authority of the Northern Territory (IHANT).
- provide professional support to Aboriginal Environmental Health Workers (EHWs).
- strengthen health promotion practice at the community level.
- conduct environmental health surveys, research and development.

Environmental Health comprises several discrete services, which includes:

- Public and Environmental Health Standards
- Aboriginal and Community Environmental Health
- Environmental Planning and Built Environment
- Food Safety
- Poisons Control
- Radiation Protection
- Water Quality

Major Achievements

Impact on Health Gains

- Continuation of the Wadeye Scabies Treatment Intervention program which is collaboration between Environmental Health, Thamarrurr Regional Council and Westmead Hospital. Prior to the introduction of the Skin Health Program in 2000, the scabies rate in Wadeye amongst children aged under 5 was as high as 33%. The program has succeeded in reducing this rate to as low as 3%.
- Remote Food Service Worker training conducted in Central Australian remote communities to improve food safety.
- Assisting Medical Entomology Branch in a comprehensive Aedes Aegypti mosquito control program in Tennant Creek.
- Advices for drinking water were issued to major indigenous remote communities due to microbiological water quality failure. These advices recommended boiling of water or use of packaged bottled water as a precaution.
- Various Dog Health programs held throughout East Arnhem. Example of benefit being at a major East Arnhem Community with a population of around 400 people and now only 48 dogs. This community has not
had any major outbreaks of diarrhoea, scabies or any other skin diseases in children during the year.

- Various health promotion/education programs carried out on East Arnhem communities, including and incorporating school screening programs.
- Investigation into Murray Valley Encephalitis case on a Central Australian remote community
- DHCS Environmental Health Program acts as a grantee for two National Aboriginal Strategy – Environmental Health Program (NAHS-EHP) projects:
  - NAHS-EHP Round 2 Septic Tank Upgrade had an initial budget of allocations of $1.65 million. Septic tank systems at Titjikala, Laramba, Attitjere and Irrelirre were completed in 2002/03. The project was expanded to include upgrade of septic tanks at Amoonguna and was completed in 2004. The funding of the project is now $2.14 million.
  - NAHS-EHP Round 2 Water Supply Upgrade project provided infrastructure to 30 remote outstations to 30 remote outstations in Jabiru, East Arnhem, and Katherine regions. This project was also expanded to include an additional 15 outstations and upgrades were completed in 2004. Current budget allocation of $3.05 million.
- Darwin Urban investigated:
  - 48 cases of suspected norovirus among diners who ate imported oysters at a local restaurant. This investigation helped instigate a nationwide food recall of the contaminated batch of oysters.
  - 10 cases of confirmed salmonellosis among guests at an event that were catered for by a local restaurant.
  - 8 cases of confirmed salmonellosis among diners (including one food handler) at a local restaurant.
  - The Alice Springs Town Council has established the framework for an EHW program.
  - Development of a Faecal & Other Body Fluid Accident Policy, which is used specifically for Lake Leanyer and commercial swimming pools.
- Disease Outbreak training:
  - OzFoodNet, in conjunction with DHCS conducted a training program on the Investigation of Gastro Illness and Outbreak Management, in Gove, Katherine, Alice Springs and Tennant Creek. The training aims to enhance skills and provide information for all staff involved in investigations of gastrointestinal illness. Staff from OzFoodNet and the Environmental Health Unit presented information on all aspects of foodborne disease investigations, and included practical exercises and workshops to enhance learning.

Poisons Control:
- S8 and Restricted S4 Clinical Advisory Committee has been established under the Poisons & Dangerous Drugs Act and met in Nov 2003 and June 2004. The function is to provide both policy and specific advice into the supply of S8 and restricted S4 substances (including opiates and amphetamines) to the Chief Health Officer.
- Pseudoephedrine Poster launched in December 2003 in conjunction with the Pharmacy Guild NT branch, NT Pharmacy Board and NT Police.
- Update of S29 notices under the Poisons & Dangerous Drugs Act for registered nurses and aboriginal health workers employed at specified remote area health centres.

Successes

- Radiation Protection Act – Bill passed 28/4/04
- Public and Environmental Health Bill - drafting instructions are being finalised by Parliamentary Counsel. The Bill will be introduced in the November sittings for debate in the February 2005 session. It is anticipated that the Act will commence mid 2005.
- Public and Environmental Health Standards-work is proceeding on subordinate regulations and standards to support the Bill. The draft Standard for Commercial Accommodation is currently being circulated
Challenges

- The entire Environmental Health Program will be involved in undertaking Community Housing Surveys on all major and minor Aboriginal communities in 2004/05 for the Indigenous Housing Authority of the Northern Territory. The information will be used to assist Indigenous Community Housing Organisations to provide timely repairs and maintenance to all community housing to improve housing functionality and beneficial health outcomes.

Improvements

- Review of Environmental Health Survey forms culminating in a Housing Survey workshop.
- Improvements with program data capture and comprehensive reporting mechanisms.
- Movement towards full program staffing

Future Directions

- Implementation of the new Food Act, Public and Environmental Health Act and Radiation Protection Act.
- A review of the Aboriginal Environmental Health Worker Program.
- Further refinement of Environmental Health activities and interventions through evidence based health policy.
- Alice Springs Town Council will be establishing an Environmental Health Worker Program.

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Diarrhoea—when it is non-infectious

Mary Verus, TB Program, CDC, Darwin

My name is Mary Verus and I have been an admin officer at the Centre for Disease Control (CDC) in Darwin for 10 ½ years. I have a condition called coeliac disease (CD) and this is my story about how my life has been affected by this disease.

On June 6 1985, I gave birth to my first child – Jesse. We had awaited Jesse’s arrival with much anticipation as my first pregnancy had ended in a miscarriage and for some reason I did not fall pregnant quickly.

Jesse died not long after his birth. His post-mortem report showed that he had a lumbosacral myelocoele, hydrocephalis and bilateral talipes. From this, I basically understood that he had spina bifida. I left the hospital a few days later, trying to understand.

It was suggested that I have a folic acid test as recent discoveries had shown that there was a connection between low folate levels in pregnancy and spina bifida. It has been too many years to remember the exact results, but I remember they were staggeringly below normal. I was told to start taking folic acid tablets daily.

Over the years I had suffered from spasmodic diarrhoea. The doctors had always passed it off as a bug and given me Lomotil to reduce it. However after Jesse’s birth I began to find the
diarrhoea much worse. Several times at the supermarket and once at a restaurant I had to be taken home because I completely lost control of my bowels. My GP thought this was probably due to my “terrible nervous state” at the time.

One day my mother went to a new GP as she was unwell and she told him my story. He said “so she has a folic acid deficiency – is she thin and does she have diarrhoea?”. Of course the answers were “yes” and I was sent to a gastroenterologist, given a biopsy and a diagnosis of CD was made.

When I look back, from about 1978, over a period of maybe 6 months, I lost approximately 12kg. From then on I suffered spasmodic diarrhoea, abdominal cramping and at one stage in about 1979 I was diagnosed with a folic acid deficiency which was treated with injections.

I am a healthy 61kg these days. I have 2 beautiful healthy children and follow a strict gluten-free diet.

Since my diagnosis several close relatives, including my brother, have been diagnosed with CD. My father died of cancer at age 31. We cannot prove that he had CD, however Mum says that he was always unwell and was not accepted into the armed forces because of poor health and his mother suffered bouts of unexplained diarrhoea.

CD is a genetically based permanent intolerance to dietary gluten (found in wheat, rye, barley and oats). This causes villous atrophy of the small bowel mucosa which in turn leads to malabsorption and a predisposition to gastrointestinal malignancy, particularly carcinoma of the oropharynx and oesophagus and small bowel lymphoma.

Why is the diagnostic rate so poor? One reason is that it can present in many guises. It has been described as – “the mantle of the great mimic of disease”.

Symptoms may include fatigue and/or chronic anaemia; diarrhoea and/or constipation; bone and joint pain or muscle spasms; ulcerations and/or swelling of mouth and tongue; gastrointestinal symptoms including flatulence, bloating, nausea and vomiting; unexplained infertility/recurrent miscarriage; unexplained early onset of osteoporosis, and tooth decay.

A number of other disorders associated with underlying CD are dermatitis herpetiformis, type I diabetes mellitus, autoimmune thyroiditis, Down’s Syndrome, epilepsy, peripheral neuropathy, autoimmune liver disease and primary biliary cirrhosis.

Recently the CD Support Group in Darwin was very lucky to have two special visitors – Graham and Cheryl Price. Graham is the technical officer for the Australian Coeliac Society and his wife, Cheryl, is the co-ordinator of the NSW Branch as well as being the editor of *The Australian Coeliac* which is the group’s national magazine. He and Cheryl jointly developed the Coeliac Society of Australia’s new ingredient list.

Some of the points highlighted by them were as follows:

- The incidence of CD is increasing. Why? Because of the introduction of blood screening tests 10 years ago. When someone is diagnosed with CD these days their immediate family is encouraged to have the blood screening and because of this more people are being diagnosed. Also more and more people are hearing about CD and taking themselves off for testing.

- CD is defined as a European/Asian Disease. There is no documentation of Aboriginal people having CD. It is not found in the Oriental Asian Community. It is believed that 50% of children in India with diarrhoea may have CD and 20% of people in Iran with diarrhoea may have CD.

- It can affect people of all ages, and is just as likely to present in those over 60 years as it is in those under 20 years.

- The general ratio of CD between women to men is 2.4 to 1. However between the ages of 20-50 the range increases to 4 to 1 which is due to the physical stress on women during their childbearing years, leading to anaemia.

- Gastrointestinal symptoms are now generally the exception to the rule as a reason for diagnosis. A card is currently being prepared which will be sent out to GPs asking that patients with unexplained anaemia or tiredness be tested for CD.
In one of the southern Fracture Clinics all people with fractures have been tested for CD. Of the people attending the clinics, it was found that 1 in 15 tested positive for CD and osteoporosis caused by CD may have contributed to the fractures.

CD can affect all of the body’s organs. A man on the Liver Transplant Program in the USA was discovered to have CD. Once he was placed on a gluten free diet, his transplant was not required.

Dr Glenn Reeves, Staff Specialist in Immunology at John Hunter and Royal Newcastle Hospitals, wrote in The Australian Coeliac dated September 2003 that CD meets all 5 criteria put forward by WHO for justifying broad-based screening approaches:
1. CD is common (around 1:100).
2. It is often silent or atypical in presentation.
3. Reliable and effective screens exist.
4. Effective treatments are available.
5. Treatment averts later morbidity and mortality.

All type 1 diabetic children get tested for CD. Undiagnosed CD can trigger type 1 diabetes, if you are susceptible.

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Norovirus detected in oyster meat
Karen Dempsey, OzFoodNet Enteric Disease Epidemiologist, CDC, Darwin

In November 2003 a large outbreak of gastroenteritis involving guests who had eaten at a popular Darwin restaurant occurred over a 2-3 week period. A cohort study design was used to investigate the outbreak and an imported brand of oyster meat was strongly implicated as the cause. The evidence included a relative risk of greater than 16 for this food item, the presence of norovirus in the stools of cases and the fact that the outbreak commenced shortly after the oyster meat was introduced onto the menu, and ceased once the product was discarded. The oyster meat, used as a substitute for the restaurant’s usual variety of partially shucked New Zealand oyster as a means of reducing the time used in the shucking process, was grilled for 8 to 10 minutes prior to serving.

The wholesaler was contacted and a large quantity of oyster meat (10kg) from the same batch obtained for testing. The testing was conducted at 2 Adelaide laboratories, but neither managed to detect norovirus in any of the samples. After much deliberation the implicated batch was eventually withdrawn from the market in March 2004, and the labeling on subsequent batches changed to highlight a warning to customers to “Cook Before Consumption”.

In June 2004 the Darwin Centre for Disease Control was notified of another outbreak of gastroenteritis linked to oyster meat consumption. Investigation of the outbreak revealed that 5 of 8 guests at a private dinner party in Darwin on 29 May had became ill with diarrhoea, vomiting, severe abdominal and muscular aches and pains within 24–48 hours of consuming raw oyster meat. The guests, assuming a plate of oyster meat placed on the dining table in preparation for cooking was edible, had consumed between 1 to 2 oysters each. Devoid of its packaging, with the warning to cook before consumption, the raw oyster meat, looked like a plate of large Australian oysters, which are considered safe to consume without cooking.
Of the 3 people who were not ill, 2 had eaten the oyster meat steamed and 1 had not eaten any. Faecal specimens were obtained from 2 cases, both of whom were convalescing at the time, and both were negative for pathogens.

The brand of oyster meat was the same as the brand, (Jiffy Brand IQF oyster meat) implicated as the cause of the November 2003 norovirus outbreak (NT Disease Control Bulletin, March 2004). It was sold directly to the public in 1kg bags (as shown in picture) by a local seafood retailer and purchased by the hostess on the morning of the dinner party.

In light of the repeat occurrence of illness among consumers of imported oyster meat, and the fact that clinical symptoms appeared to be consistent with norovirus, we decided to test the oysters for the pathogen, this time using a laboratory (the Institute of Environmental Science and Research Limited, in Porirua, New Zealand) with a proven methodology for detecting viruses in oysters.

A 1 kg bag, presumed to belong to the same batch as that consumed on 29 May, was collected from the retailer and arrangements made to freight it to the laboratory. Coincidently, we also decided to send over another sample, a 1kg bag of oyster meat leftover from last year’s outbreak. This bag had been stored in the freezer, still sealed, at the environmental health unit since December 2003.

**Results**

Norovirus was not detected in the sample implicated in the most recent outbreak. This result was not entirely surprising considering that norovirus had not been detected in any of the stools of cases. We may have wrongly assumed that this bag belonged to the batch eaten by the dinner guests, and it may have belonged to a new batch delivered in the few days prior to collection.

In contrast, the left over bag tested positive for norovirus. This was a surprising outcome because even though epidemiological evidence had strongly implicated the oysters, the chance of detecting norovirus was considered low in light of the 2 Adelaide laboratory results. In addition, prolonged freezing was thought to adversely affect the test’s ability to detect norovirus (personal communication, Allamanda Faatoese). Yet, despite 6 months of freezing, a detectable amount of norovirus remained, suggesting gross contamination prior to harvest or at processing.

**Discussion**

Two issues were raised from these outbreaks. Firstly, they illustrated that it remains very difficult to get microbiological evidence of norovirus contamination of food. For this reason the Australian Quarantine and Inspection Service (AQIS), which expends considerable effort constraining the importation of contaminated foods, does not routinely test for viral pathogens. In the case of the 2003 outbreak, the apparent absence of norovirus in the oyster meat had the effect of delaying public health action. The eventual withdrawal of the implicated batch did not occur until much later, when in all likelihood, there was very little left in the market place.

Secondly, enhancing the “cook before consumption” warning on the oyster meat label did little to prevent further illness. In Australia raw oysters are considered an edible delicacy irrespective of their origin or means of production. Changing the behaviour of the consumer will require more than just intensive labeling.
Enteric diseases in the Northern Territory
April – June 2004
Karen Dempsey, OzFoodNet Epidemiologist, CDC, Darwin

Outbreaks

One outbreak of foodborne disease and 1 outbreak of non-foodborne disease occurred in the Darwin region during the April to June quarter of 2004.

On 7 June an investigation was initiated following a report of gastroenteritis among guests who attended a private dinner party in Darwin on 29 May. Five people became ill following consumption of raw imported Japanese oysters (see this issue p37 for full report).

On 17 May 2004 a health service co-ordinator reported an outbreak of gastroenteritis among a group of Brisbane health professionals staying at a fishing resort. The group of 9 had flown to the resort on Friday 14 May and all had been well until the early hours of Sunday 16 May when 1 of the group reported gastro-enteric symptoms. A further 7 reported similar symptoms over the ensuing 24 hours. Local community health staff and a Darwin-based environmental health officer conducted an outbreak investigation, interviewing all guests using standard questionnaires and collecting faecal specimens from cases.

Although an inspection of the resort premises revealed no major breaches in food hygiene and safety, several frozen food items exceeded recommended storage time and were discarded. Three possible exposures were investigated; food eaten at the resort, food eaten on the flight from Brisbane and water consumed at the resort. Food consumption at the resort was investigated and found to be identical among those who were ill and those who were not. In addition there were no ill foodhandlers nor were there major breaches in food safety identified in the kitchen. QANTAS food quality personnel were contacted to determine whether other passengers had reported illness following the flight. There were no reports of illness associated with that flight, nor were there any reports of illness among passengers who had traveled on other flights that day but had consumed the same food. Finally, water was considered a possible exposure as the supply had failed to pass routine testing on the day prior to onset of illness. This possibility was ruled out, as residents at the nearby community drinking the same water supply were unaffected.

In summary the investigation failed to link the source of infection to the resort or the plane flights. Two of the faecal specimens tested positive for norovirus and transmission was presumed to be person-to-person.

Clusters

Cluster surveillance is conducted on a weekly basis by monitoring trends in notifiable diseases data looking for groups of enteric disease cases with common characteristics such as serotype and age or location, and by comparing current 4 weekly counts with historic counts (line listing).

During the 2nd quarter this surveillance detected a cluster of 11 Shigella flexneri 2A cases over 3 weeks. Most were residents of Alice Springs and of various ages, ranging from 1 to 57 years. This was unusual because even though S. flexneri is frequently reported in residents of remote Central Australian communities it is far less common in urban residents. The investigation failed to identify links to food or water, however there was some evidence of person-to-person transmission within families.

A second investigation was initiated after examination of the Northern Territory Notifiable Diseases System identified a cluster of cases of Salmonella Saintpaul among primary school aged children living in the city or rural outskirts of Darwin. S. Saintpaul is frequently reported in children aged less than 5 years but rarely in primary school age children. The investigation failed to identify links to food or water, however there was some evidence of animal-to-person transmission as 5 out of the 6 cases had pets including blue tongue lizards and pet mice or had handled geckos.
**Workshops**

Two Investigation of Gastrointestinal Illness Workshops were conducted in the NT in this quarter, the first in Nhulunbuy on 14-15 April and the second in Katherine on 27-28 May. Both workshops were well attended with medical staff from public health units and Aboriginal Medical Services, environmental health officers from Department of Health & Community Services and Defence Department, plus Masters of Applied Epidemiology scholars. The workshops are designed to:

1. strengthen communication networks between public health staff and environmental health officer;
2. enhance their knowledge of food borne pathogens;
3. provide skills to enable the identification and management of sporadic cases, clusters and foodborne illness outbreak investigations; and
4. perform epidemiological analysis using manual calculations and EpiInfo version 3.2.2

**Enteric disease notifications**

**Campylobacter**

During the period April to June 2004 there were 42 notifications of *Campylobacter* infection reported in the Northern Territory, far fewer than notified last quarter (63) and substantially less than the mean number (68) reported for the same period of the previous 4 years, 2000-2003. The reason for this decline is not known. The median age of 13 years also altered from the normal median age (1 to 2 years) with older children and adults accounting for 50% of all cases reported this quarter.

**Salmonella**

A total of 104 cases of salmonellosis occurred during this quarter, slightly less than the previous quarter (118) but within normal limits for this time of year. Although the majority of cases were infants and babies children aged less than 2 years of age (77%), the median age of 6 was higher than normal (generally the median age is between 1 to 2 years) suggesting sources of infection other than food or environment. This was borne out by the clustering of cases among primary school aged children for whom direct contact with animals was thought to be the mode of transmission. *Salmonella* Ball was the leading serovar (14 cases), closely followed by *Salmonella* Saintpaul with 12 cases, both of which are generally considered environmental serovars. There were no food-borne disease outbreaks reported this quarter, consistent with the decline in *Salmonella* Typhimurium notifications, (only 3 this quarter).

Two cases of *Salmonella* Enteritidis and 4 cases of *Salmonella* Paratyphi B Var Java were notified.

The cases of *S. Enteritidis* were young adults from Darwin. One was a 26 year old male with no travel history and thought to be locally acquired as a result of occupational exposure to animal faeces. The other, a 24 year old female could not be contacted for enhanced surveillance. She tested positive for *Salmonella* Enteritidis PT6A, a phage type commonly found in Bali.

The *S. Paratyphi* cases were from Darwin city (1), Katherine region (2) and Alice Springs region (1). The regional cases were 2 Indigenous infants and a 16 year old non-Indigenous male, none of whom had history of travel or contact with imported tropical fish. The Darwin case, a 20 year old male was not contactable for follow-up.

**Shigella**

Although there was a substantial increase in notifications of *Shigella* infection reported this quarter compared to last quarter (36 versus 25) the count was only slightly higher than the mean number (31) reported during the same quarter for the previous 4 years, 2000-2003. The median age (24 years) was much higher than normal with at least 50% of cases occurring in people aged over 20 years.

**Other enteric disease notifications**

Only 26 cases of cryptosporidiosis were reported this quarter, substantially less than reported during the same period in previous years. *Cryptosporidium* has been an infrequent cause of enteric disease in 2004, even during the wet season when this pathogen is most prolific.
Similarly, rotaviral notifications were well below normal with only 21 cases notified this quarter. The average count for this time of year (April to June) generally exceeds 200 cases.

There were 2 cases of hepatitis A reported, 1 in Alice Springs region and 1 in Darwin region, and no reports of sporadic haemolytic uraemic syndrome (HUS), listeriosis or yersiniosis.

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Letter to the Editor

Enteric diseases in the Northern Territory January – March 2004

We wish to respond to the article in June 2004 The Northern Territory Disease Control Bulletin entitled “Enteric diseases in the Northern Territory January – March 2004”. This article suggests that there are high rates of nosocomial transmission of rotavirus in Alice Springs Hospital.

We reviewed all cases of rotavirus reported by the Alice Springs Hospital laboratory during January 2004. There were 74 cases of rotavirus diagnosed in January by the hospital laboratory.

Three of these cases were neonates in Special Care Nursery who had never been outside hospital, and clearly their infections were nosocomial. One of these cases had returned to Alice Springs Hospital from the Women’s and Children’s Hospital in Adelaide, but it is likely that the infection was acquired in Alice Springs.

One child was admitted with gastroenteritis, but without rotavirus isolated. This child was discharged then re-admitted with rotavirus infection within the 3 day incubation period of rotavirus. One long-term inpatient acquired the infection in hospital. Thus there are at least 5 definite cases of nosocomial transmission.

We are concerned that there has been this amount of nosocomial rotavirus infection, though this is less than the rate you have reported. Our data do not include patients who could have been infected with rotavirus while in Alice Springs Hospital, then discharged, and did not return to the hospital.

We are undertaking research to better understand the level of nosocomial rotavirus infection and to enable appropriate action. In particular we are investigating the use of passive immunisation with a rotavirus antibody in all paediatric patients who do not have rotavirus during outbreaks. We shall report on the effectiveness of this in the future.

Yours sincerely,

Dr Rosalie Schultz
Public Health Medical Officer
Centre for Disease Control
Alice Springs

Michelle Callard
Infection Control Nurse
Alice Springs Hospital

Dr Rob Roseby
Acting Head of Paediatrics
Alice Springs Hospital

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Notifications in the 2nd quarter 2004 compared to the mean of the 2nd quarter for the previous 4 years: selected diseases*

*Those diseases with less than 5 notifications in the 2nd quarter 2004 were excluded unless of public health significance. Please note that the quarterly count for legionellosis was significantly different from previous years however was excluded from the graph as there were only 2 cases.

Notifications in the 2nd quarter 2004 compared to the mean of the 2nd quarter for the previous 4 years: sexually transmitted infections and blood borne viruses*.

*Excludes gonococcal ophthalmic neonatal as there were 0 notifications.
## NT Notifications of Diseases by Onset Date & Districts
1 April to 30 June 2004 and 2003

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<th>Darwin</th>
<th>East Arnhem</th>
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<td>0</td>
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<td>775</td>
<td>45</td>
<td>54</td>
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</table>
Adverse vaccine reaction

The large number of vaccinations given in the Meningococcal C School Program has resulted in a small increase in adverse events after immunisation reported in this quarter.

Hepatitis C

Increasing hepatitis C notifications may be the result of greater practitioner awareness following concentrated efforts by the Department of Health and Community Services and non government organisations to improve access to screening and treatment for populations at risk of hepatitis C. Current information available on cases of hepatitis C does not allow the differentiation of newly acquired infections from those that are not. This makes it difficult to determine if a rise in cases is indicative of an increase in transmission of hepatitis C among those at risk, or long term carriers presenting for testing.

NT Malaria notifications January – March 2004

Merv Fairley, CDC, Darwin

Eight notifications of malaria were received for the 2nd quarter of 2004. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

<table>
<thead>
<tr>
<th>Number of cases</th>
<th>Origin of infection</th>
<th>Reason exposed</th>
<th>Agent</th>
<th>Chemoprophylaxis</th>
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<tbody>
<tr>
<td>1</td>
<td>PNG</td>
<td>holiday</td>
<td>P. vivax</td>
<td>yes</td>
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<tr>
<td>2</td>
<td>Sudan</td>
<td>refugee</td>
<td>P. falciparum</td>
<td>yes</td>
</tr>
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<td>1</td>
<td>East Timor</td>
<td>working</td>
<td>P. falciparum</td>
<td>no</td>
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<td>1</td>
<td>East Timor</td>
<td>working</td>
<td>P. falciparum</td>
<td>yes</td>
</tr>
<tr>
<td>2</td>
<td>Uganda</td>
<td>refugee</td>
<td>P. falciparum</td>
<td>no</td>
</tr>
<tr>
<td>1</td>
<td>Indonesia</td>
<td>unknown</td>
<td>P. falciparum</td>
<td>no</td>
</tr>
</tbody>
</table>

HTLV-1

HTLV-1 most commonly occurs in Aboriginal people in the Central Australian region and is not screened for routinely. There were 12 notifications this quarter, slightly higher than the average reported during the previous 4 years, however interpretation is difficult with such small numbers.

Neisseria Gonorrhoea

The notifications for genital gonorrhoea are higher than usual for this time of year when compared with the average number of notifications for the same period in the previous 4 years. Central and Top End jurisdictions of the NT have undertaken extensive community STI screening programs this year which is likely to have impacted on the detection of a greater number of cases. An increase in gonorrhoea cases is, however, a trend reflected nationally.
Disease Control staff updates

AIDS/STD Program

Katie Rosewarne commenced as Clinic 34 receptionist, previously having worked for RFDS at Ayers Rock. Maggi Richardson has taken short term leave with Gabrielle Bennett filling her position. Alison Males, recently employed as a locum for sexual health will be working on the Syphilis Information System in Alice Springs.

CDC—General

Keshini Richards has resigned from the Alice Springs assistant co-ordinator position, moving to Canberra and Coleen Doherty has been employed in this position for 3 months. A 6 week position is being filled by Nicci Douglas assisting with general administration and supporting Environmental Health. The Rheumatic Heart Disease Program coordinator position in Alice Springs has been vacated by Beth Rowan.

Environmental Health

Alice Springs EHOs, Lisa Sutton and Clayton Doyle have resigned to take similar positions in Queensland. Ray Anderson (Katherine) and Russell Spargo (Darwin) have completed their short term contracts and returned to environmental health work in Victoria.

Immunisation

Christine Selvey, NT Head of Immunisation, has taken a senior public health position with the Victorian Dept of Human Services until July 2005. Tania Wallace has returned to CDC to take up the Immunisation Section Head for this time period.

Medical Entomology Branch (MEB)

Raelene Whitters has commenced with MEB in a new position as Data and Information Officer. Matthew Shortus has been employed in Darwin while Gisela Lamche is on maternity leave and Bill Pettit will be working on the Tennant Creek mosquito eradication program.

TB/Leprosy

Noela Davies has transferred to the public health nurse position in Tennant Creek while John Turahui is on 3 months leave. Noela has recently been working in Ethiopia on a health and water project.

Eileen Jones AM

The recent death of Eileen Jones at the age of 75 marks a milestone in the history of public health in the Northern Territory (NT).

Eileen had devoted her working life to the eradication of leprosy in the NT after arriving here in 1952.

She initially worked in Katherine and Darwin hospitals, mainly in the area of midwifery.

In 1957 Eileen joined the Aboriginal Affairs Welfare Branch and went to work at Lajamanu. She was transferred to Maningrida in 1958 where she arrived by ship with the building materials for her house. During this time she developed her interest in leprosy.

Back in Darwin in 1963, Eileen worked as a technical officer at East Arm Leprosarium and then as the first full time leprosy control field officer in the NT with the Leprosy Control Unit, Darwin. She taught many remote nurses and medical officers about leprosy in her visits.

Eileen co-authored the book *Leprosy in Tropical Australia* (1984) with Dr John Hargrave, taking many of the photographs that illustrate the text.

During her years in Leprosy Control Eileen visited communities throughout the NT screening for leprosy and reviewing patients. She was known far and wide in Aboriginal communities and cattle stations out the Territory and her recollection of her patients and their families was amazing. She was in her element while travelling, with her love of the bush and her many friendships developed over the years.

Eileen was made a Member of the Order of Australia in 1988 and retired in 1994 after 40 years of service.