THE NORTHERN TERRITORY

DISEASE CONTROL BULLETIN

ISSN 1440-883X Vol. 6, No. 2, June 1999

Meningococcal disease in Central Australia 1998

Belinda Farmer, CDC, Alice Springs

From June to December of 1998 Central Australia experienced an outbreak of meningococcal disease. Most of the cases were Group B. The increase of cases coincided with the commencement of the winter months and persisted to the end of the year. During this time there was an increase in respiratory disease with influenza A and Respiratory Syncytial Virus (RSV) being the viruses predominantly detected.

Method

Meningococcal disease cases are notified to Alice Springs Centre for Disease Control (CDC) usually via the Alice Springs Hospital (ASH) laboratory. Cases up to June were those fitting the definitions for confirmed and probable as per Table 1. By June/July 1998 it was evident that the region was experiencing an outbreak and surveillance was enhanced. The case definition was broadened to include suspect cases (Table 1) and detection of meningococcal antigen in the blood as per the National Health and Medical Research Council (NH&MRC) Guidelines for the Control of Meningococcal Disease in Australia1.[1]

A meningococcal alert outlining the signs and symptoms of meningococcal disease and emergency management advice was distributed to ASH and all urban and remote clinics. This alert was later reissued in August 1998 by CDC Darwin, again highlighting the signs and symptoms of the disease along with the information that several NT cases had presented with non specific flu-like symptoms.

Contents

EDITOR Vicki Krause PRODUCTION DESIGN Sue Reid
ASSISTANT EDITORS Sue Reid, Frank Bowden, Christine Selvey, Tania Wallace, Paul Kelly, Merv Fairley
Chris Nagy
PRINTED Publications Unit, Territory Health Services

Centre for Disease Control, Block 4, Royal Darwin Hospital
PO Box 40596, CASUARINA NT 0811 Phone (08) 8922 8044 Fax (08) 8922 8310
E. Mail: vicki.krause@health.nt.gov.au
Website: http://www.nt.gov.au/nths/publich/cdc/bulletin.htm

Table 1 Case definitions

1. **Confirmed**[2]
   - ⇒ isolation of *N. meningitidis* from a normally sterile site eg blood, CSF, or less commonly, joint, pleural, or pericardial fluid OR
   - ⇒ detection of Gram negative intracelullar diplococci in blood, CSF or skin lesion OR
   - ⇒ isolation of *N. meningitidis* from a skin lesion in the absence of positive blood cultures

2. **Probable**[2]
   - ⇒ clinical purpura fulminans in the absence of positive blood cultures: OR
   - ⇒ detection of meningococcal antigen in CSF (useful as a surveillance tool but limited in use for individual management)

Note: Positive antigen test results from urine or blood samples are unreliable for diagnosing meningococcal disease.

3. **Suspect**[3]

   If the CSF Gram stain, culture and latex agglutination tests for *N. meningitidis* are negative but the clinical and CSF findings are consistent with pyogenic meningitis (leukocytes $>1x10^6$/L, polymorphs >75%) and at least one case has been confirmed in the patient's community at about the same time.

Specific directions were given with regard to taking blood cultures whenever possible, and a nasopharyngeal swab or throat swab prior to administration of antibiotics.[4]

CDC Alice Springs alerted any new cases to CDC Darwin and the Director of the Public Health Unit Alice Springs.

Contact tracing was commenced as early as possible by community staff, with CDC staff assisting when requested. Close contacts were defined according to the NT guidelines[2] (see Table 2), although this definition was somewhat arbitrarily applied because of travel between communities and Alice Springs. In communities who had Aboriginal Health Workers (AHWs), defining a contact was made much easier because of their local and family knowledge. CDC organised preventive treatment for all contacts of all cases. Although rifampicin (10/mg/kg/dose orally 12 hourly for 2 days) was the drug of choice, some communities, after consultation with AHWs and the contacts themselves, preferred to use the single dose of ceftriaxone for compliance reasons. The dosage schedule used for ceftriaxone was:

Weight ≤ 25kg: 125 mg dissolved in 1% lignocaine hydrochloride, as a single intramuscular dose.

Weight > 25kg: 250 mg dissolved in 1% lignocaine hydrochloride, as a single intramuscular dose.

Table 2

A contact is defined as:

1. Anyone who has spent:

4 hours or more each day for 5 consecutive days, or more than 24 hours in total with the index case in the week preceding the onset of illness.

2. Anyone who has had significant contact with oral secretions of the index case during the 10 days preceding onset of disease, ie mouth-kissing, or any staff member who performed mouth-to-mouth resuscitation.

Results

There were 11 confirmed cases, 2 probable and 1 suspect as shown in Figure 1. The one suspected case had a weakly reactive *N. meningitidis* polymerase chain reaction (PCR) result. All cases but one were Aboriginal. There was only one death in a case who was continuously hospitalised post treatment and died several months after with meningococcal disease as a contributing factor. Five out of the 11 confirmed cases exhibited a rash.

Figure 1 Reported cases of meningococcal disease in Central Australia 1998

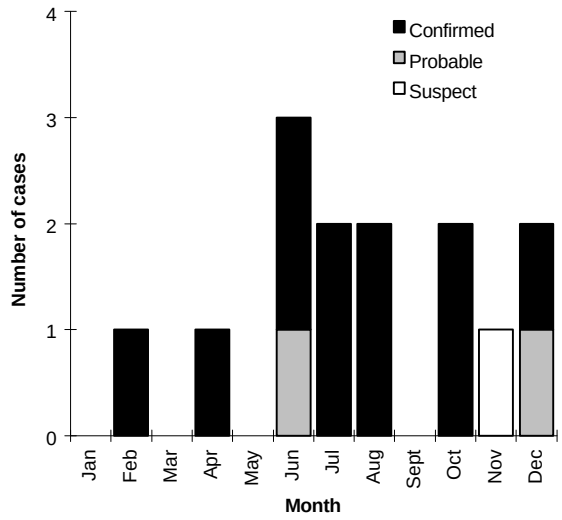


Table 3 shows the demographic information on the meningococcal cases notified to CDC, Alice Springs during 1998. Most cases were in the 0 to 5 year old age group (n=7). Four cases were between 19 to 29 years and 2 cases were over 50 years of age. Eight had received antibiotics prior to septic workup. Four cases received nose swabs prior to admission of which 3 were culture positive.

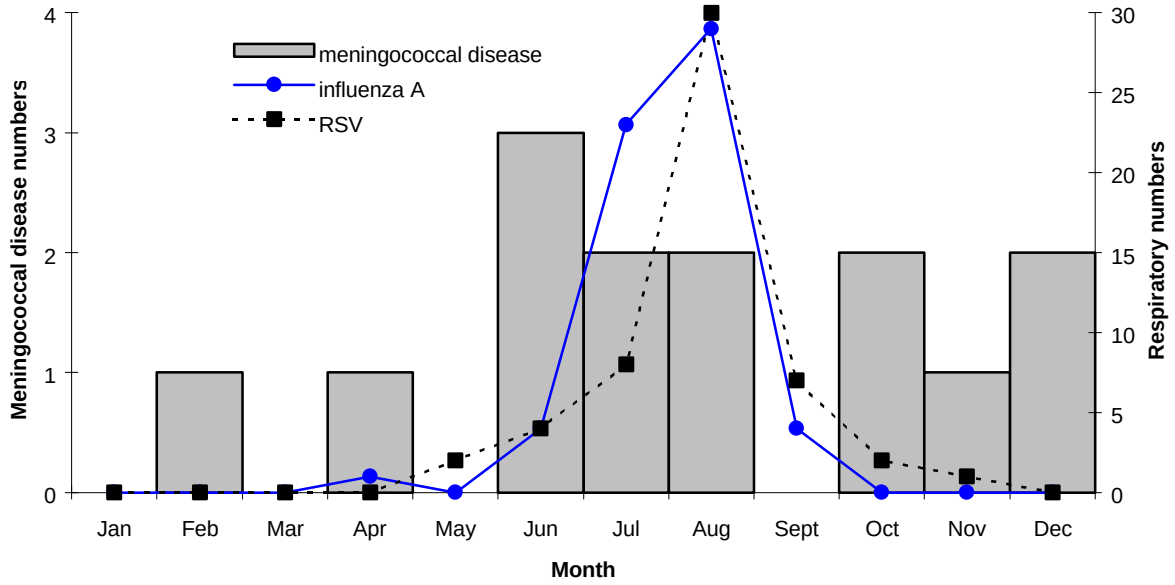
In June/July 1998 Central Australia also was noted to have an increase in respiratory illness both in communities and in the urban area. In 1998, influenza was not notifiable in the NT, so evidence to support this increase in disease was obtained from diagnosis of Influenza A and RSV made at the Institute for Medical and Veterinary Science on specimens referred from ASH (Figure 2). A preceding respiratory illness was described in 11 of the 14 cases.

Table 3 Demographic information on cases notified as meningococcal disease to CDC Alice Springs, 1998

Onset	Clinical Picture	Community	Sex	Ethnicity	Age in years	Culture group/other	Sero/sub type	Rash
14/02/98	Meningitis	Urban	F	Aboriginal	1.5	B	2b:N/ST	Purpura fulminans
09/04/98	Septicaemia	Remote 1	F	Aboriginal	65	B	2b:N/ST	No
03/06/98	Septicaemia	Remote 2	F	Aboriginal	4	C	2a:P1.5	"Fading rash"
14/06/98	Meningitis	Urban	M	Non Aboriginal	54	B	2b:N/ST	Petechiae
22/06/98	Meningitis/ pneumonia	Remote 3	M	Aboriginal	22	Nil/*		No
01/07/98	Meningitis/ pneumonia	Remote 4	F	Aboriginal	0.8	B	NT:N/ST	No
26/07/98	Meningitis/ pneumonia	Urban	M	Aboriginal	5.6	Nil/gram neg intracellular diplococci*		Petechiae
08/08/98	Septicaemia/ bronchitis	Remote 5	M	Aboriginal	0.7	B	NT:1.5	Petechiae
08/08/98	Meningitis/ pneumonia	Remote 4	F	Aboriginal	3	B	2b:N/ST	No
16/10/98	Meningitis	Remote 6	M	Aboriginal	0.1	B	2b:N/ST	No
27/10/98	Meningitis	Urban	M	Aboriginal	1.9	B	NT:N/ST	No
07/11/98	Meningitis	Remote 4	F	Aboriginal	11	Nil/#		No
15/12/98	Meningitis	Remote 4	M	Aboriginal	28	Nil/†		No
29/12/98	Septicaemia/ endocarditis	Urban	F	Aboriginal	20	C	2a:N/ST	No

NT = nontypeable * Blood Latex B # PCR positive
N/ST = non subtypeable † CSF Latex ACY,W135 positive

Figure 2 Reported number for all ages of meningococcal disease and respiratory illness in Central Australia 1998



Discussion

Central Australia has seen major epidemics of meningococcal disease disproportionately affecting Aboriginal people between 1971-1973, and again between 1987-1991.[3] The 77 cases of meningococcal disease diagnosed over the five years 1987-1991 were mainly serogroup A; sixty of these cases were classified as confirmed, seven probable and ten suspected.

This is the first time Central Australia has had a meningococcal outbreak of predominantly Group B. Most cases were in children under the age of 5 years and young adults. No secondary cases were identified. Two cases had family links with index cases who had previous disease within a 81 and 130 day time frame. These cases had not received prophylaxis, and were not defined as contacts according to the definition which requires specified contact hours with the index case within the week preceding onset of illness.[5]

At the time a clinically suspected case is reported to CDC, not all the laboratory investigations are available, and the early intervention with antibiotics may preclude obtaining evidence to support a confirmed diagnosis. During this 'waiting' time, there were 6 cases whose diagnosis was clinically compatible with meningitis/septicaemia. One case had a positive urine latex test, three others had a meningitis with raised CSF polymorph counts and two others had clinical meningitis. With the increase in confirmed disease locally and the need for prompt public health action it was decided locally to contact trace and offer preventive treatment for these cases. This action was accepted and carried out in the scenario of increased incidence of a life threatening disease but did create a large work load, increased distribution of antibiotics and concern in the community.

A blood culture prior to antibiotics is important to isolate a microorganism. Where a blood culture has not been taken a nasopharyngeal swab may be helpful. In this series of cases a nasopharyngeal/or throat swab was not routinely collected but should be considered as a possible addition to the NT Meningococcal Guidelines in the future.

The NH&MRC and NT Meningococcal Guidelines do not include the detection of meningococcal antigen from urine in diagnosing meningococcal disease, though the NH&MRC do accept antigen detection in blood for notification and this is supported in an outbreak situation.[6] Although PCR became available in November 1998, the time delay in Central Australia for obtaining results makes it an unsuitable indicator for urgent public health intervention.

During this increased activity of meningococcal disease, Central Australia also was experiencing an increase in respiratory illness. A high proportion of the meningococcal cases had a preceding respiratory

illness, often diagnosed and treated as pneumonia, or presumed to be a flu-like illness. An association with influenza and meningococcal disease has been previously documented[7] and heightened surveillance for meningococcal disease is needed in this scenario.

Acknowledgments

Special acknowledgment goes the health care staff who managed the clients, to Sandra Thompson who collected much of the information and also to Angela Merianos, Fran Morey, Vicki Krause and Sue Reid for their help and expertise.

References

1. National Health and Medical Research Council. Guidelines for the control of meningococcal disease in Australia. Canberra: AGPS, 1997.
2. Territory Heath Services, Disease Control Program. Guidelines for Meningococcal Meningitis/Septicaemia Chemoprophylaxis. December 1997.
3. Patel M, Merianos A, Hanna JN, et al. Epidemic meningococcal meningitis in Central Australia 1987-1991. *Med J Aust* 1993; 158: 336-340.
4. Merianos A. Centre for Disease Control, Darwin. Meningococcal Disease Memo/Alert. August 1998.
5. Stuart JM, Cartwright AV, Robinson PM, et al. Does eradication of meningococcal carriage in household contacts prevent secondary case of meningococcal disease? *BMJ* 1989; 298: 569-570.
6. American Academy of Pediatrics. Meningococcal infections. In : Peter G, ed 1997 Red Book: Report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village, IL: American Academy of Pediatrics; 1997: 357-362.
7. Cartwright KAV, Jones DM, Smith AJ, et al. Influenza A and meningococcal disease. *Lancet* 1991; 338:554-557.

****

Editorial

That meningococcal disease can progress to a life threatening illness within hours of first symptoms underscores the urgency for early diagnosis, prompt treatment and prevention of the disease where possible.

The above article by Farmer reminds us that we are in the peak season for meningococcal disease, ie winter (The Dry) and the season continues through early spring. Heightened awareness for the disease is needed.

Central Australia has a history of meningococcal outbreaks[1] and it is interesting that all of the 1998 NT notified cases were from the Centre with 12 of the 14 cases occurring from June to December. The disease is most common in 0-5 year olds, teenagers and young adults and the elderly and all the cases were in one of these groups.

Early recognition that several meningococcal cases were occurring specifically within the Central Australian Aboriginal population raised the possibility of clusters in communities and highlighted the need to obtain cultures for group typing. To guide disease control measures typing is needed to establish whether the outbreak is potentially vaccine preventable (ie disease from serogroups A, C, and W135) and having confirming culture results will support contact tracing and the administering of preventative treatment to eliminate nasopharyngeal carriage in asymptomatic contacts. This elimination will prevent ongoing transmission and further invasive disease. Being almost exclusively a Group B outbreak precluded vaccination but the decision to broaden the case definition ie to include suspect cases and blood antigen positive compatible clinical cases in this outbreak setting was sound and allowed for possibly early diagnoses and also contact tracing in the one case. Going beyond these extended case definitions may be understandable due to fear of the disease but may hinder focussing on those truly at risk, lead to widespread use of antibiotics and cause undue alarm. Health care providers and the public need to know everything possible is being done in accordance with best practice and health care providers involved with the cases and the communities need to have access to up to date local and national guidelines.[2]

Health care providers are urged to get blood cultures from cases prior to antibiotics where possible but not to unduly delay treatment. Nasopharyngeal swabs should be routinely collected and the organism can often be cultured from this site despite previous antibiotics. This is clearly indicated in the NH&MRC guidelines[3] but is not in the NT guidelines[4] and should be added. The finding of a positive nasopharyngeal culture with clinically compatible disease could also be considered in the 'probable' case definition. The aspiration of purpuric lesions for laboratory confirmation can also be useful, again, particularly in the setting where

antibiotics have been given before blood or CSF cultures have been taken.[2]

Finally, as has been noted, meningococcal disease has been documented to follow outbreaks of respiratory disease, specifically influenza A and therefore a further preventive measure can include high level influenza vaccination coverage in those at risk.[5,6,7,8] This may provide protection at least to the elderly at risk group.

The above outbreak and shift to serogroup B in the Centre adds more impetus to the ongoing work to develop a safe and effective meningococcal Group B vaccine.

References

1. Patel M, Merianos A, Hanna JN, et al. Epidemic meningococcal meningitis in Central Australia 1987-1991. *Med J Aust* 1993; 158: 336-340.
2. O'Grady KA, Krause V. Report cases on clinical suspicion, consider skin-lesion films/aspirates for diagnosis [letter]. *Med J Aust* 1998; 168: 195-196.
3. Territory Heath Services, Disease Control Program. Guidelines for Meningococcal Meningitis/Septicaemia Chemoprophylaxis. December 1997.
4. National Health and Medical Research Council. Guidelines for the control of meningococcal disease in Australia. Canberra: AGPS, 1997.
5. Krasinski K, Nelson JD, Butler S, Luby JP, Kusmiesz H. Possible association of *Mycoplasma* and viral respiratory infections with bacterial meningitis. *Am J Epidemiol* 1987; 125: 499-508.
6. Cartwright KAV, Jones DM, Smith AJ, et al. Influenza A and meningococcal disease. *Lancet* 1991; 338:554-557.
7. Young LS, LaForce FM, Head JJ, Feeley JC, Bennett JV. A simultaneous outbreak of meningococcal and influenza infections. *N Engl J Med* 1972; 287: 5-9.
8. Moore PS, Hierholzer J, DeWitt W, et al. Respiratory viruses and mycoplasma as cofactors for epidemic group A meningococcal meningitis. *JAMA* 1990; 264:1271-1275.

Two cases of Hib in a Central Australian community

Jenny Hains, CDC, Alice Springs

Case 1

On 15 January 1999 a 2 year old male Aboriginal child, was admitted to Alice Springs Hospital (ASH) with fever, vomiting and diarrhoea. The child had been evacuated from a nearby community, having been treated with IM penicillin. A septic screen yielded abnormal CSF, with positive latex for *Haemophilus influenzae* type b (Hib). The child was commenced on dexamethasone and cefotaxime. The initial blood culture subsequently grew Hib.

The case was notified to CDC, Alice Springs by ASH laboratory on 17 January 1999. CDC staff initiated an investigation of the child's immunisation history and contact tracing in accordance with *The Australian Immunisation Handbook*[1] recommended guidelines. Examination of the NT Childhood Immunisation Database and the child's community health clinic records confirmed the administration of three doses of PRP-OMP (PedvaxHIB) vaccine. The child was age appropriately immunised, having received 3 doses of PRP-OMP vaccine in a timely fashion from three different community health centres. Only one batch number had been recorded and that was for the second dose of PRP-OMP. The child's immunisation history was otherwise complete.

Case 2

On 6 February 1999, a second male Aboriginal child aged 9 months from the same community as Case 1 was admitted to ASH with diarrhoea cough and fever. He had had a recent admission one week earlier with diagnosis of bronchiolitis, left lower lobe pneumonia, gastroenteritis and iron deficiency anaemia. Blood cultures performed on that admission were negative.

A septic screen was performed including CXR, blood culture, urine and CSF. Blood culture grew Hib; the CSF was normal. The had initially been treated with IV penicillin for his respiratory illness. This was changed to cefotaxime once blood culture results were known.

On 9 February 1999, CDC was notified of a positive Hib blood culture from the child. With the guidance of Alice Springs CDC staff, contact tracing in accordance with the recommended guidelines[1] took place within the community. Contacts in the hospital included two boarding mothers and another child.

The child's immunisation history was examined and revealed a total of four PRP-OMP immunisations recorded as given at 2 months of age, 4 months of age, 4.5 months of age and 6 months of age (the 3rd and 4th dose are not in accordance with the recommended schedule which is to have a 3rd and final dose at 12 months). Three of the Hib injections were given at the same health centre and one was given in ASH. The first two Hib vaccinations did not have the batch numbers recorded against them. The remaining immunisation history was complete.

Response to apparent vaccine failure

The two Hib isolates have been sent from ASH laboratory to Sydney for further typing. Following consultation with the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) and consistent with recommendations of the American Academy of Paediatrics[2], the two children were offered a booster dose of PRP-OMP. Four weeks after the booster, serum will be collected from the index cases and sent to a reference laboratory for analysis of antibody levels. The appropriate Hib vaccine failure questionnaires were completed and sent to NCIRS.

A reminder letter was sent to all Central Australian vaccine providers reiterating the importance of recording the batch numbers of vaccines given in the event of vaccine failures requiring investigation.

Acknowledgement

Special thanks to Dr Anne Jaquiery from the Paediatric Department (ASH) for her assistance.

References

1. National Health and Medical Research Council. The Australian Immunisation Handbook. 6th ed. Canberra: AGPS, 1997: 102-109.
2. American Academy of Pediatrics. *Haemophilus influenzae* infections. In: Peter G, ed. 1997 Red Book: Report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village, IL: American Academy of Pediatrics; 1997: 220-231.

****

Editorial

Prior to the introduction of *Haemophilus influenzae* type b (Hib) conjugate vaccines for children 0-5 years in 1993, rates of invasive Hib disease in the Northern Territory (NT) were high (incidence rates of 141/100,000 with 24 cases/year on average)[1] with Central Australian Aboriginal children having the highest rates ever reported. Vaccines which conjugate the Hib polysaccharide to carrier proteins have been very successful in producing protective antibodies in young infants and children making Hib disease now a rare occurrence. In 1995, 1996, 1997 and 1998 there were 4, 3, 3 and 0 cases reported respectively in children 0-5 years.

As the NT and the nation work towards the elimination of Hib, it is important to monitor and evaluate all cases of Hib disease to assess vaccination program effectiveness and the effects on Hib carriage. This will require a sensitive and reactive surveillance system and the laboratory capability to serotype all suspected isolates. Recently, Dr Peter McIntyre of the National Centre for Immunisation Research and Surveillance (NCIRS) of Vaccine Preventable Diseases, has raised several issues regarding the surveillance of Hib disease and evaluating the infrequent vaccine failures. These include:

1. The need to monitor all *Haemophilus influenzae* invasive disease (HI), ie Hib and non-Hib, to pick up any trends in increases of non type b invasive disease as Hib disease declines.
2. The importance of accurate serotyping to confirm that cases truly are type b, especially when laboratories may no longer be keeping up-to-date kits for typing HI. Organisms from immunised cases must be confirmed to be type b before they can be "true" vaccine failures. Typing will show any non Hib trends as per point 1 above and will also detect any shift to other capsular types of Hib over time.
3. The need for demographic and risk factor information for cases of vaccine failure. This is presently collated via the Australia Paediatric Surveillance Unit (APSU). However there is a need for specific steps of action in dealing with a suspected case of Hib disease, eg:

    i. If the child was not eligible for immunisation (ie younger than 2 months or older than 5 years in 1993):

- Forward isolate for confirmatory typing to designated laboratory. No further investigation.

ii. If the child is a potential vaccine failure (two or more doses given in the first year of life, one or more doses given over 12 months of age):
   - Forward isolate for confirmatory typing to designated laboratory.
   - Endeavour to save the first available blood sample (preferably serum) after hospital admission for later antibody testing.
   - If a child has not been age appropriately immunised for Hib, complete the course.
   - If the child has been age appropriately immunised for Hib then either repeat Hib antibody testing in 2 to 4 weeks after recovery (to assess whether the child has responded to the Hib polysaccharide following infection) and immunise if no significant antibody response is seen (a follow up test to show response to the conjugate vaccine may then be considered) or just give another dose of Hib vaccine and measure Hib antibody 2 to 4 weeks after immunisation.

iii. In either case the APSU form (or similar updated form including risk factors) should be filled out and forwarded to the NCIRS.

It should be remembered that contacts of Hib cases should be followed up as per the current immunisation guidelines.[2]

The above points have been presented to the Communicable Diseases Network of Australia & New Zealand (CDNANZ) to identify the roles that Public Health Units/Departments and the CDNANZ play in Hib surveillance. To have a sensitive surveillance system collaboration is needed among the "coal face" health care providers, the laboratories, the Public Health Units/Departments, the CDNANZ, the APSU and the NCIRS.

Already a study from an Alaskan Native population with similar pre-vaccination rates of Hib disease to that of NT Aboriginal children has shown that Hib vaccination did not reduce or eliminate Hib oropharyngeal carriage. Hib cases increased in this population with a change to a vaccine which provides a more gradual increase to protective Hib antibody levels.[3] Carriage studies in the NT post-vaccination have also shown a return to pre-vaccination Hib carriage (personal communication Amanda Leach). This emphasises that there is no room for complacency and high levels of Hib vaccine coverage must be maintained. Sensitive surveillance will help identify areas of continuing transmission and detect populations in need of improved vaccine coverage.

References

1. Markey P. The effect of conjugate Hib vaccines on the incidence of invasive Hib disease in the NT. *NT Comm Dis Bull* 1998; 5(1): 3-6.
2. National Health and Medical Research Council (NHMRC). The Australian Immunisation Handbook. 6th ed. Australian Government Publishing Service: Canberra, 1997.
3. Galil K, Singleton R, Levine OS, et al. Reemergence of invasive *Haemophilus influenzae* type b disease in a well-vaccinated population in remote Alaska. *J Infect Dis* 1999; 179: 101-106.

"Unvaccinated child dies of *Haemophilus influenza* type b infection"

This was the title of the leading article in CDR - Communicable Disease Report Weekly from the UK on 7 May 1999. The article tells of a 6 year old girl from Trent region (UK) who acquired epiglottitis while holidaying in Spain. She was hospitalised and given ceftriaxone but died the following day. *Haemophilus influenza* type b (Hib) was cultured from blood. She had not been vaccinated.

The NT is working towards 100% coverage with Hib vaccination to avoid such preventable deaths.

Outbreak of hepatitis A in a Darwin urban primary school

Jacki Mein, CDC, Darwin and MAE program, NCEPH, ANU, Canberra

Introduction

On 25 November 1998, the Centre for Disease Control (CDC), Darwin received a Royal Darwin Hospital notification of serologically confirmed hepatitis A in a seven year old girl attending a local primary school. Her onset of illness had been 15 November. On 27 November a general practitioner rang CDC having seen two children with vomiting and jaundice from the same primary school and the following day serology confirmed hepatitis A in both. All three were confined to one class area of the school. The school invited CDC to investigate.

Aims of the investigation

To prevent further spread of the infection, both in the immediate and longer terms.

- To seek any possible common source for the cases.
- To actively look for other, unidentified cases of hepatitis A at the school.
- To offer immune globulin to contacts of confirmed cases of hepatitis A.
- To promote good preventive hygiene practices - hand washing after toileting and before eating.
- To assess canteen and toilet facilities and general hygiene.

Methods

A case definition was developed, a site visit was performed, and information obtained initially by structured questionnaire and supplemented by telephone interviews. Age, sex, ethnicity, residential address, household details, food handling, contact with child care, attendance at gatherings where food was consumed, and travel history were all recorded via the CDC Top End Enteric Investigation form. Contacts were defined as household members or sexual contacts of cases. As the children in the class area were toilet trained and were not supposed to share food except in a supervised class context they were not defined as contacts.

A proven case was defined as a positive hepatitis A IgM, and a probable case defined as having jaundice and bilirubinuria in a child/staff member from same class area of the primary school during the two weeks 15 November to 1 December.7

Interventions

Recently exposed family members of cases were contacted and those with no known history of hepatitis A were identified and offered immunoglobulin (serologic testing not performed).

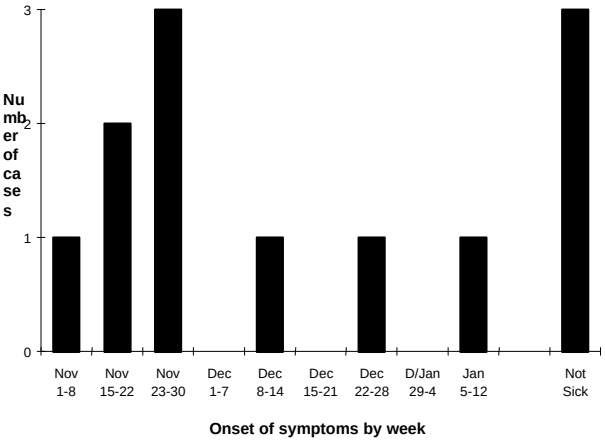
Parents, principal and staff of the class area were provided with oral and written information on prevention of further disease spread. CDC wrote announcements for the weekly school newsletter to all parents and the school directed queries from all concerned parents and staff to CDC.

An environmental health officer performed a school assessment of foodhandling practices and toilets. Standards were acceptable though minor modifications were recommended.

Results

Of the 12 cases, 6 were identified initially as 5 children from 2 grades and 1 teacher in the same class area who became sick within two weeks of each other. Another three occurred as a result of secondary transmission, one to two months later. Three further cases were asymptomatic, identified retrospectively as contacts of secondary cases so their time of onset was unknown (see Figure).

Figure Outbreak of hepatitis A in a Darwin primary school, 1998/99 (n=12)



The median time between onset of symptoms and diagnosis was 6.5 days (range, 3-11).

The crude attack rate for clinical illness in the class area (including 6 staff working daily in the class area over the preceding months) was 6/48 or 12.5%. Amongst pupils it was 5/42 or 11.9%. Asymptomatic seroconversion occurred in at least one child at the primary school and two children attending the preschool.

Of eligible contacts without a history of hepatitis A, 15/17 (86.7%) accepted and received immune globulin. Median time from diagnosis to treatment of non-immune contacts was one day (range 0-4) with the exception of the parents from the first case who refused treatment until another case occurred in their family four weeks after the first. No contact had received a full course of hepatitis A vaccine previously. Interestingly 3/22 (13.6%) contacts did not require immune globulin because of a history of past disease.

No clear common source was found, in food, drink, play or contact histories. The teachers did not remember any event where younger siblings were changed on tables shared by the classes, or both classes participated in any cordial/drink or food sharing, finger/face painting, or swimming lessons. Other possible sources included regular combined classes with the preschool and a local special school, but neither the children or their supervisors were clinically unwell, though not serologically tested. Three cases attended after school care but the supervisors both tested negative and there were no linked cases.

Discussion

Food borne outbreaks of hepatitis A usually have an abrupt onset with a high number of cases in a short time, rather than the slow rise and prolonged fall of case numbers in the more common person to person transmission. A wide spectrum of people can be infected in food-borne outbreaks, compared to the usual person to person groups, ie children under 5, and young adults, where spread is faecal-oral.[1] The children and teacher affected and the close spacing of our cases in time and place suggests a common source. Only three later cases of person to person transmission were confirmed serologically, although with many contacts being younger, potentially asymptomatic children, further transmission within young families possibly occurred.

It seems unlikely that one infected person was responsible for directly infecting others. An infectious case probably contaminated food or drink. To date no common source has been definitively identified for this outbreak. Whatever occurred was probably during the period of the last two weeks of October or the first week of November in the shared class area.

The diagnosed cases of hepatitis A may have been only a small percentage of the actual number of cases, as not all possible cases were tested. This was because asymptomatic or mild infection, did not prompt parents to seek testing, and because of the lack of a non-invasive test to use in young children.

Morbidity

Two children were hospitalised for three days, the first with vomiting for investigation, the other with a seizure the day after diagnosis. The fit was thought by the treating doctors to be unrelated, although there is an isolated report of meningo-encephalitis following acute hepatitis A.[2] The teacher had two weeks off work. Two other children spent four days off school, apart from the recommended exclusion period. This necessitated time off for working parents.

Outbreak containment - local

Could we have prevented any cases of hepatitis A in this outbreak? By the time CDC was notified, the 6 probable point source cases were already symptomatic or late in the incubation period. A further 3 were asymptomatic and were picked up as a result of testing contacts of secondary cases.

Two of the three secondary cases occurred in adults living in households with well children from the same class area. The third case occurred late in school holidays in a child from the class area who had initially tested negative for hepatitis A. He had had no contact with other school children during the holidays. However, his brother attended the preschool and on testing was also positive for hepatitis A. It is postulated that his brother was infected asymptomatically around the time of the outbreak, who then infected him some weeks later.

If immune globulin had been given routinely to all children in the class area, as it would have been in the USA[3] or Queensland,[4] the 2 adult secondary cases would not have been prevented as the children who passed on the infection were probably already infected, and there is no evidence that immune globulin prevents excretion of hepatitis A.[5]

Conclusion

An outbreak of Hepatitis A is difficult to effectively investigate in the absence of classic symptoms such as jaundice, in a young population with asymptomatic infections, concurrent gastrointestinal infections and lack of a non-invasive specific test. For the same reasons it is difficult to control, as evidenced by at least three secondary cases. Although the first cases were most likely the result of a common source transmission, no causative event or food was defined. Control by vaccination was not indicated for contacts because of current lack of evidence on both cost effectiveness, and post exposure vaccine efficacy in an outbreak setting.

Outbreaks elsewhere - the evidence and methods for interventions

Although timely immune globulin is effective at preventing cases in household contacts,[6] it does not halt person to person spread of disease within a community. Often outbreaks are protracted and difficult to control until the pool of susceptible persons is exhausted.[7] While mass immunoglobulin administration has been trialed and does appear to have some success in contained populations where high rates of coverage are achieved[4,8] it is still questionable whether containment occurs because of the restricted nature of a population rather than purely as a result of immunoglobulin administration.

There is no direct charge for immunoglobulin. However the costs of administering it ie time, expertise and consumables - mean it is an expensive part of an intervention.[9]

It is unclear whether the hepatitis A vaccine is effective if given post exposure in a foodborne outbreak setting, though there is now some evidence that vaccinating household contacts of sporadic cases with a single dose of vaccine may prevent some secondary cases.[10] In addition, there is some evidence that unlike immune globulin the vaccine decreases excretion of hepatitis A.[11] If hepatitis A vaccine administration was considered instead of or in conjunction with immune globulin to control an outbreak, (with the vaccine cost varying between $35 and $70) it would markedly increase the intervention cost. There have been calls for routine vaccination of school children in the USA to prevent transmission in the young[12] although there are concerns about the long term implications of waning immunity post immunisation.

In our setting and on the evidence available at the time we elected not to give vaccine, and offered immune globulin to eligible contacts. However in light of the above, further randomised studies looking at costs of intervention and efficacy of vaccine in outbreaks are needed, and recommendations for outbreak management may alter in the next few years.

Acknowledgments

Grateful thanks to Mark Myerscough from Casuarina Night and Day Clinic, and Sue Reid, Merv Fairley, and Vicki Krause from CDC Darwin for fieldwork, editing assistance and comments.

References

1. Mosley WH, Speers JF, Chin DY. Epidemiologic studies of a large urban outbreak of infectious hepatitis. *Am J Public Health* 1963; 53: 1603-17.
2. Matsushima K, Niwa K Fujita H, Yamamoto M, Shinohara Y. Acute hepatitis A presenting findings (sic) of meningoencephalitis. Rinsho- Shinkeigaku 1992; 32(4): 441-3 (abstract only - non English publication).
3. Centres for Disease Control and Prevention. Prevention of hepatitis A through active or passive immunisation: recommendations of the Advisory Committee on Immunisation Practices (ACIP) *MMWR Morb Mortal Wkly Rep* 1996; 45:1-30.
4. Hanna, JN. Hepatitis A in a child care centre. *Comm Dis Intell* 1993; 17: 73-5.
5. Thompson SC, Le Leu LA. Don't forget the hepatitis A vaccine. *Med J Aust* 1997; 167: 228-9.
6. Stapleton JT. Passive immunisation against hepatitis A. *Vaccine* 1992; 10: S45-7.
7. Pavia AT, Nielsen L, Armington L, et al. A community-wide outbreak of hepatitis A in a religious community: impact of mass administration of immune globulin. *Am J Epidemiol* 1990; 131: 1085-93.
8. Prikaszky V, Olear V, Cernoch A, et al. Interruption of an outbreak of hepatitis A in two villages by vaccination. *J Med Virol* 1994; 44: 457-9.
9. Dalton CB, Haddix A Hoffman RE, Mast EE. The cost of a food-borne outbreak of Hepatitis A in Denver, Colo. *Arch Intern Med* 1996; 156: 1013-6.
10. Sagilocca Luciano, Pietro Amaroso, Tomaso Stroffolini, et al. Efficacy of hepatitis A vaccine in prevention of secondary hepatitis A infection: a randomised trial. *Lancet* 1999; 359:1136-9.
11. Innis B, Snitbahn R, Kunasol P. Protection against hepatitis A by an inactivated vaccine. *JAMA* 1994; 271:1328-1334.
12. Koff RS. The case for routine childhood vaccination against hepatitis A. *N Engl J Med* 1999; 340: 644-5.

Brief reports - outbreaks in April and May 1999

Chris Evans, CDC Darwin and MAE Program, NCEPH, ANU, Canberra

Gastroenteritis

In April 1999, a point source outbreak of acute gastroenteritis occurred in a tour group visiting Kakadu National Park. Thirteen of 17 people (attack rate 76.5%) became ill within a 24 hour period with nausea, vomiting, diarrhoea and upset stomach being the major symptoms. The outbreak was brought to the attention of the Centre for Disease Control (CDC) staff in Darwin by the Urban Environmental Health team who received a complaint from a member of the tour group. Several of the group had complained to the NT Tourist Commission.

CDC arranged for food histories to be collected from those members of the tour who had not dispersed and additional information eg onset and duration of symptoms was obtained via an emailed questionnaire. Fifty percent of the questionnaires emailed were returned, indicating a high level of motivation by the group to determine the cause of the outbreak.

A particular water supply was implicated as the source of the infection by a number of the group, which included an overseas lawyer with experience in environmental health and water boards. Testing of the water did not support bacterial pathogens as the cause of disease and onset times of illness of 1 to 16 hours following ingestion did not support a theory of heavy metal contamination. Site inspections conducted by Environmental Health Officers revealed no major breaches of hygiene or food storage practices and there were no suspicious environmental conditions.

There were no reports of gastrointestinal illness in members of two concurrent tours with the same caterer and similar itineraries.

One member of the tour group required medical treatment at Royal Darwin Hospital (RDH) Accident and Emergency Department, however no faecal samples were collected. No faecal samples were obtained from any of the group and no food was available for testing. The incubation period for the disease was 1-2 days and illness duration was less than a week, suggesting a viral pathogen. It was reported that one member of the tour had been ill with a gastrointestinal illness prior to the trip commencing, however details of the illness could not be obtained. No causative organism was identified, nor was a particular food or water source implicated by the investigation.

Measles

A cluster of measles cases was reported in Darwin in May 1999. The first, in a two year old unvaccinated boy, was reported to the Centre for Disease Control (CDC) Darwin on 7 May 1999. A second serologically confirmed case in a 25 year old male was notified via the laboratory system on 12 May. A further three cases, all unvaccinated siblings of the two year old, were reported in the following three weeks.

There have been no further reports of measles in the three weeks since onset of illness in the last notified case.

The first case had 20 identifiable contacts that included siblings, parents, co-workers of the mother, a pathology courier and patients in the waiting room of the general practitioner who made the diagnosis. Isolation of 10 contacts for varying periods was requested by CDC staff for those of uncertain measles immunity. The unvaccinated siblings of the child developed measles within 24 days of their brother. As they had been isolated from school and from public contact for several days prior to the onset of illness they did not pose a risk for dissemination of measles to the community.

The second case had 10 close contacts, four of whom were health care workers in the Paediatrics and Accident and Emergency Departments of Royal Darwin Hospital (RDH). The hospital staff were followed up by the RDH Infection Control Nursing Director. Other contacts included infants in the general practitioner's waiting room too young to have received measles, mumps and rubella (MMR) vaccine. The risk of infection for individual infants was assessed and parents informed of the possibility of exposure and subsequent infection. The time lapse between possible exposure and notification of the case by the laboratory meant that prophylactic immunoglobulin could not be offered.

Although there was no direct contact between the first and second cases, their activities at the probable time

of transmission suggest a common measles contact, most likely a national or international traveller. The child had been a constant passenger on an airconditioned bus carrying air travellers at the time and the young adult had spent time in a Darwin internet cafe frequented by travellers.

This cluster follows an interstate traveller-borne outbreak of measles earlier this year. Onset of rash, fever and cough in travellers should continue to be viewed with a high index of suspicion for measles. Suspected cases should be notified to CDC promptly and isolated as appropriate, and contacts identified and offered vaccination according to their immune status. Travellers about to embark on overseas trips should be encouraged to have an MMR, especially those in the vulnerable age group of 18 to 40 years who may not have had natural measles, missed vaccination or had a suboptimal vaccine preparation.

Non-Communicable Diseases Update: No.6. Message: Prescribe moderate physical activity

Tarun Weeramanthri, Community Physician, CDC, Darwin

Did you know that...?

A. Physical activity has a wide range of health benefits

Physical activity reduces the risk of developing coronary heart disease, hypertension, colon cancer, and diabetes.[1] It enhances mental health; fosters healthy muscles, bones and joints; and helps maintain function and preserve independence in older adults.

B. Accumulating moderate intensity activity is highly beneficial - vigorous activity is not necessary

Significant health benefits can be obtained by including a moderate amount of physical activity (eg 30 minutes) on most, preferably all, days of the week.[1] Moderate activities include brisk walking, gardening and swimming - activities that increase your heart rate but do not necessarily make you puff. This amount of activity can be accumulated over the course of a day. It does not have to be taken 'in one hit'. Previously, vigorous physical activity involving large muscle groups and lasting at least 20 minutes on 3 or more days per week was recommended.[2]

C. Physical inactivity is a powerful and common cardiovascular risk factor

The level of decreased risk of coronary heart disease attributable to regular physical activity is similar to that of other lifestyle factors, such as not smoking.[1]

From a population perspective, physical inactivity is important because it is so common (more common than hypertension and smoking for example). Booth[2] estimated that 50% of the adult Australian population are insufficiently active. Women are less likely to be active than men, and older people are less active than younger adults.

D. Getting the sedentary moving conveys great benefits

From a population perspective, getting the most sedentary to increase their activity levels (even a little) will result in large health gains.[2] The benefits of activity have been shown in those aged over 60 years, even in the group who have been sedentary for most of their lives. Of course, people who are already physically active will also benefit by increasing the intensity or duration of their activity.

E. Physical activity should be prescribed

The intensity, duration and frequency of physical activity should be prescribed like a medication wherever possible. The prescription should be individualised and take into account the particular circumstances of the patient's life. An Australian study asked the physically inactive to identify their preferred activities and barriers to participation.[3] The most preferred activity was walking and the most frequently cited barriers to more regular participation were insufficient time, lack of motivation and child care responsibilities (in the youngest age group), and injury or poor health (in the oldest age group). The exercise prescription should be recorded and progress monitored at a subsequent visit.

It should be acknowledged, however, that the intervention is not straightforward and more research is needed on the feasibility, efficacy and efficiency of

all lifestyle interventions in a general practice setting.[4,5] Exercise prescription may be suitably delivered as a brief intervention technique.[6]

Policy makers also have a key role in changing the physical and social environment to make it more conducive to physical activity.[7] Educational and environmental interventions should be seen as complementary.[7]

Active Australia is a national initiative to promote physical activity. Its slogan is 'Exercise. You only have to take it regularly, not seriously.' For more information you can view its website at www.ausport.gov.au/partic/activeoz.html, or phone Steve Morton on 8922 8280 at the Chronic Diseases Network office for more information.

References

1. US Surgeon General. Physical Activity and Health: A Report of the Surgeon General. Atlanta: US Department of Health and Human Services, and Centers for Disease Control and Prevention, and President's Council on Physical Fitness and Sports, 1996.
2. Booth ML. Physical activity: what's afoot? *Aust NZ J Public Health* [editorial] 1997; 21: 557-558.
3. Booth ML, Bauman A, Owen N, Gore CJ. Physical activity preferences, preferred sources of assistance, and perceived barriers to increased activity among physically inactive Australians. *Prev Med* 1997; 26: 131-137.
4. Little P, Margetts B. The importance of diet and physical activity in the treatment of conditions managed in general practice. *Br J Gen Pract* 1996; 46: 187-92.
5. Ashenden R, Silagy C, Weller D. A systematic review of the effectiveness of promoting lifestyle change in general practice. *Family Practice* 1997; 14: 160-176.
6. Brown K. Physical activity: a new message for general practice. *The Echo* (Top End Division of General Practice Newsletter) 1999; January: 1.
7. Sallis JF, Bauman A, Pratt M. Environmental and policy interventions to promote physical activity. *Am J Prev Med* 1998; 15: 379-97.

An update on leprosy control in the NT

CDC, Darwin

As leprosy moves toward elimination, the priority of activities within leprosy control in Territory Health Services (THS) is being evaluated and changed. Recently TB/Leprosy staff of CDC, THS discussed realistic approaches to service delivery and leprosy control. Yesteryear, the focus of leprosy control was on continual surveillance and recall of individuals with the disease, of those suspected of disease and of leprosy contacts. Today, given the very low incidence of the disease and with more knowledge about the natural history of the disease a different approach is required.

The initial classification of leprosy cases is very important as a classification determines infectivity, prognosis, disease complications, treatment regimes and protocols for follow up of contacts. There are two commonly used classifications for leprosy. The first is the Ridley and Jopling system, with a progression from the mildest from of disease to the more disseminated disease:

TT, Tuberculoid → BT, Borderline Tuberculoid → BB, Borderline → BL, Borderline Lepromatous → LL, Lepromatous

The second is the WHO Classification which is based on both the clinical picture and the results of skin smears. **Multibacillary leprosy includes any patient with positive skin smears**, and generally includes those at the more "disseminated" end of the spectrum ie some BT, BB, BL, LL. **Paucibacillary leprosy includes those patients with negative skin smears** and generally includes those with more limited clinical disease ie TT, most BT.

This classification has been developed for countries which use skin smears for diagnosis, but do not necessarily have access to biopsies, as we do in Australia. Our Guidelines for Leprosy Control in the NT have previously included the results of AFB's found on biopsy to classify patients, and thus determine follow up, however we are now adhering more strictly to the WHO definition using skin smear results only. Thus all diagnosed leprosy clients require annual clinical review, and only those who are skin smear positive (and not biopsy smear positive), require annual skin smears.

Contact follow up can be extremely time consuming and frustrating at times, given the insidious nature of the disease. The recommended follow up period is determined by the incubation period of leprosy. The

present 20 year follow up is generous, and still remains the optimum, however, flexibility needs to be used. For example if a contact was seen only once at 18 years post exposure, he or she may be discharged. If a contact is never reviewed, an effort should be made to review the person once, but if unsuccessful the individual should be deemed "lost to follow up". It is very important however to have clear documentation on patients' records in hospital or at the community where a leprosy patient/suspect or contact has lived. This is important for keeping up the awareness of the disease in this period of decreased prevalence.

Finally just to remind people that the leprosy ELISA test is no longer recommended (or available).

Leprosy elimination moves a step nearer

(Source: World Health Organization Internet site: http://www.who.int/inf-pr-1999/en/pr99-25.html)

According to the World Health Organization (WHO) leprosy is nearing elimination worldwide as a public health problem. However, at the end of 2000, there may still be about 10 countries where the leprosy burden is greater than WHO's target level of less than one case per 10 000 population.[1] Since 1985, the use of multidrug therapy (MDT) has already reduced the global prevalence of the disease by 85%, and the number of countries with more than one case per 10 000 has dropped from 122 to only 28 at the start of this year.

Today, over 9 million leprosy patients have been cured, with extremely low relapse rates, and some 800 000 patients are registered for treatment globally, compared to more than 5 million in 1985. Since 1995, WHO has distributed the three MDT drugs to more than 4 million patients living in 71 endemic countries.

WHO estimates that nearly 2 million people have yet to be detected over the next two to three years. Of these, 90% live in 13 countries of Africa, the Americas and Asia, where, at the beginning of 1999, the prevalence rate was still 4.4 per 10 000 population. The 13 recognised at present as "top endemic countries" are, in order of prevalence, India, Brazil, Indonesia, Myanmar (Burma), Madagascar, Nigeria, Mozambique, Nepal, Ethiopia, Democratic Republic of Congo, Niger, Guinea and Cambodia.

Reference

1. World Health Assembly Resolution WHA44.9 in 1991 committed Member States to the goal of reducing the global prevalence of leprosy to less than one case per 10 000 population by the year 2000.

Lyssavirus prevention strategy update

Nan Miller, CDC, Darwin

A new lyssavirus, first identified in 1996, has been found in several species of flying foxes and bats in Australia. It has been provisionally named Australian bat lyssavirus (ABL). It is closely related to, but not identical to classical rabies virus. On-going serological testing and virus studies on bats suggest that the lyssavirus is widely distributed in Australia. It should therefore be assumed that *all* Australian bats have the potential to carry this lyssavirus.

Recommendations for prevention of lyssavirus infection in people are updated as more information becomes available. The first update was reported in the *NT Communicable Diseases Bulletin* Vol. 4, No 4, December 1997.

The National recommendations were updated in October 1998 and again in May 1999. The changes include: site and method of administration of rabies immunoglobulin (RIG); timing (to wait or not to wait) of post-exposure prophylaxis; and timing for pre-exposure booster doses.

Current post exposure prophylaxis (PEP) recommendations for ABL

- ***Immunoglobulin***

RIG should be given as a single dose at the same time as the first dose of the post-exposure vaccination course. RIG must not be given at the same *site* as the vaccine. The dose is 20 International Units per kilogram of body weight.

As much of this dose, as possible, should be injected in and around the wound site or proximal vicinity. If vaccination has commenced more than seven days prior, RIG should not be administered.

- ***Vaccination***

  Post exposure vaccination consists of five doses of 1ml rabies vaccine given as an intramuscular injection on days 0, 3, 7, 14 and 28. Doses should be administered in the deltoid area or, in children, the anterolateral aspect of the thigh is also acceptable.

- ***Timing for PEP***

  The decision as to whether PEP should be started immediately or delayed depends on: previous history of rabies vaccination in the exposed person; the severity of the bite; and the availability of the bat for testing.

1. PEP should commence immediately if:

- a person has been bitten severely by a bat, **or**
- the bat appeared to be behaving abnormally, **or**
- the bite was unprovoked (eg, the person made no attempt to handle the bat), **or**
- if results of tests to determine the presence of lyssavirus infection in the bat cannot be obtained within 48 hours of exposure, **or**
- further delays in PEP administration are anticipated, **or**
- the bat is not available for testing.

  Every effort should be made to test the bat immediately for evidence of infection, if possible to do so without endangering other individuals.

2. PEP may be delayed for up to 48 hours if the bat can be tested for evidence of lyssavirus infection and the exposed person has been:

- previously immunised against rabies; **or**
- only scratched by the bat; **or**
- bitten by the bat, **and**
- the bite was single/minor and provoked; **and**
- the bat was not obviously behaving abnormally; **and**
- the bat is available for testing, **and**
- results of lyssavirus infection in a bat can be obtained within 48 hours of exposure, **and**
- PEP can be commenced, if indicated, immediately afterwards.

If there is evidence of lyssavirus infection in the bat, full PEP should be completed.

If there is no evidence of lyssavirus infection in the bat, then PEP can be discontinued, unless the person will have ongoing exposure to bats and has not completed a full pre-exposure vaccination course.

The following table shows the number of persons who received PEP in the NT in 1998.

Table Post-exposure treatments for lyssavirus in the NT in 1998

	Vaccine only	Vaccine plus RIG	RIG only	Treatment unknown
No. of persons	3	7	0	0

Current pre-exposure prophylaxis recommendations for ABL

- ***Vaccination***

Pre-exposure vaccination should be recommended to those whose occupation puts them at risk of being bitten or scratched by bats (eg bat carers, wildlife officers, veterinary laboratory staff) and consists of three doses of 1ml rabies vaccine on days 0, 7 and 28.

- ***Booster vaccination***

Booster doses of rabies vaccine should be considered for immunised persons who have ongoing exposure to bats in the following manner:

1. Persons who work with live bat lyssavirus (eg veterinary laboratory staff) require

- blood test for rabies antibody titre every 6 months **and**
- booster with one dose of rabies vaccine if titre is less than 0.6 IU/ml.

2. Persons who come into contact with bats (eg bat carers, wildlife officers) require

- blood test for rabies antibody titre every 2 years **and**
- booster with one dose of rabies vaccine if titre is less than 0.6 IU/ml.

Remember, the best protection against bat lyssavirus is to **NOT HANDLE BATS** and to call Parks and Wildlife on 8999 4536 if you are concerned about a distressed bat. If a bat scratch or bite is incurred seek medical attention immediately.

For more detailed information on these changes and PEP, refer to the following flow chart or contact the Centre for Disease Control in your district.

Adapted from the recommendations of the Lyssavirus Expert Group and endorsed by the Communicable Diseases Network Australia New Zealand, 1998.

NT School Age Hepatitis B Program

The NT School Age Hepatitis B Program was officially completed on April 30 1999. This program offered free hepatitis B vaccination to all NT children between the ages of 6-16 years.

Over 41,000 hepatitis B vaccines were given during the 12 month campaign. The vaccines were administered in urban areas by Community Care Centre staff mainly at schools and in Community Care Centres, and in rural and remote areas at health clinics by their staff. The success of the program in delivering so many vaccinations throughout the Territory is due to the commitment and hard work of all rural and urban clinic staff who carried out this substantial program in addition to their usual work, and to the two NT Program Coordinators.

Some children have received only 1 or 2 doses of the 3 dose vaccination course within the 'official' time frame of the program which ended on 30 April 1999. These children **can** and should complete their hepatitis B vaccination course under the program. Vaccine for this purpose can be ordered from THS Regional Pharmacies by stating "School Age Paediatric Hepatitis B" on the order form.

The completion of the NT School Age Hepatitis B program means that all NT children 0-16 years have been offered hepatitis B vaccine. With coverage estimated to approach 70%, the NT is now well placed to prevent future hepatitis B transmission amongst the high risk adolescent and young adult populations. A full report on the NT School Age Hepatitis B program will be available from CDC at the end of June 1999, and a summary will be published in the next edition of the *Bulletin*.

Conference on Injury Prevention and Control - Brisbane, May 1999

Jacki Mein, CDC, Darwin and MAE program, NCEPH, ANU, Canberra

Injury prevention is a relatively new field in public health, but it is an important one. Injuries are the principal cause of death in people under 45 year of age and a leading cause of mortality, morbidity and permanent disability in Australia.[1] The prevention of injuries is highly intersectoral in nature and the mechanisms for specific injury areas are often the responsibility of non-health agencies and multiple stakeholders are frequently involved. This is why injury prevention has been designated as one of five National Health priority areas.

I attended the Third National Conference on Injury Prevention and Control held in Brisbane, 9-12 May 1999. Approximately 450 people attended, with 50 of these from countries including Canada, UK, USA, Japan, and Sweden. The delegate list was fascinating for the wide variety of groups represented- they were not just health bodies like Public health units and hospitals. Groups included data collection bodies such as the Australian Institute of Health and Welfare and ABS, emergency services like Trauma Units, Fire Departments and Police, as well as more diverse areas- Road Safety Authorities, Royal Life Saving Societies, insurance companies, coroners offices, Farmsafe, Kidsafe and surface engineering company delegates all rubbed shoulders at the conference. This was partly the solution to the problem posed as the conference theme: 'The Challenge of Integration'. Many points of view were expressed -and discussed from very different standpoints!

Highlights included a discussion on disabilities post injury and how poorly information on them is collected (Professor Lex Frieden); an exposition on selected injuries in America, how their rates differ

geographically and possible reasons why (Professor Jess Kraus), and a paper entitled "Some said a fence around the cliff, others an ambulance in the valley" aptly encompassing a discussion on the relative merits of prevention versus treatment. Other areas of public health could learn from the approach of this relatively young field.

Reference

1. Commonwealth Department of Health and Family Services and Australian Institute of Health and Welfare 1998. National Health Priority Areas Report: Injury Prevention and Control 1997 AIHW Cat No PHE 3. Canberra: DHFS and AIHW.

Keeping Track of Good Health
A series of videos for Aboriginal and Torres Strait Islander women

Access to health services and the information provided can be a positive step in improving the health status of families and individuals. Providing health information and increasing awareness of women's health screening and services are the main aims of the series of videos developed by the Women's Cancer Prevention Program. Susan Kay, Promotions Officer with the Women's Cancer Prevention Program said there are a number of reasons why Indigenous women may not be having health screening including Pap smears.

"Many feel uncomfortable or embarrassed about having women's health screening. Some don't think they are at risk of developing women's health problems and others are hampered by lack of social support or don't know where to go for a culturally sensitive consultation.

Regular women's health screening can prevent major health problems. Last year in Australia, cervical cancer screening prevented more than 700 cancers of the cervix. But three out of every four women who develop cancer of the cervix have never had a Pap smear or not had one recently."

The videos are specifically made for Indigenous women in the NT and cover such issues as Pap smears, breast screening and well women's screening.

Produced as part of the National Cervical Screening Program and the BreastScreen Australia programs in the NT, the videos will help to increase the number of Indigenous women having Pap smears, screening mammograms and well women's checks by explaining the rationale for screening and answering a range of questions commonly raised by women.

Copies will be distributed widely throughout the NT and are available from Women's Cancer Prevention Program at Territory Health Services by phoning 13 15 56.

For further information contact Susan Kay, Promotions Officer, Women's Cancer Prevention Program on 8922 5504.

NT MALARIA NOTIFICATIONS
January to March 1999

Merv Fairley, CDC, Darwin

Three notifications of malaria were received for the first quarter of 1999. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

ORIGIN OF INFECTION	REASON EXPOSED	AGENT	CHEMOPROPHY-LAXIS	COMMENTS
Indonesia	Resident	*P.falciparum*	No	Diagnosed RDH.
Indonesia	Resident	*P.falciparum*	No	Diagnosed RDH
SE Asia/India	Holiday	*P falciparum*	Yes	Diagnosed RDH

NT NOTIFICATIONS OF DISEASES BY DISTRICTS
1 JANUARY TO 31 MARCH 1999 AND 1998

DISEASES	ALICE SPRINGS		BARKLY		DARWIN		EAST ARNHEM		KATHERINE		TOTAL	
	'99	'98	'99	'98	'99	'98	'99	'98	'99	'98	'99	'98
Acute Rheumatic Fever	1	1	2	1	2	3	0	0	1	3	6	8
Adverse Vaccine React.	0	0	0	0	0	0	0	1	0	0	0	1
Arbovirus infections												
Barmah Forest Virus	0	1	0	0	8	1	2	1	2	1	12	4
Dengue	0	0	0	0	5	1	0	0	0	0	5	1
Ross River Virus	0	1	6	1	85	43	6	10	10	15	107	70
Campylobacter	26	15	0	0	41	23	2	5	5	8	74	51
Chlamydia	71	69	3	6	63	77	21	26	23	23	181	201
Congenital Syphilis	0	2	0	0	0	0	0	0	0	0	0	2
Cryptosporidiosis	21	0	0	2	4	0	0	0	7	1	32	3
Donovanosis		11	0	0	0	1	0	0	0	0	1	12
Glomerulonephritis	1		0	0	0	1	0	0	3	0	3	8
Gonococcal Disease	0	7	0	0	0	1	0	0				
Gonococcal Conjunct.	124	73	4	15	87	83	32	52	57	37	304	260
Haemophilus Inf type b	1	1	0	0	3	0	0	0	23	11	27	12
Hepatitis A	2	0	0	0	0	0	0	0	0	0	2	0
Hepatitis B	0	0	1	0	12	7	1	2	0	3	14	12
Hepatitis C (prevalence)	0	0	2	1	3	0	0	0	2	3	7	4
HIV infections	9	8	0	0	80	69	1	1	3	3	93	81
HTLV-1	0	0	0	0	1	0	0	0	1	0	2	0
Legionnaires Disease	6	14	3	0	0	0	0	0	0	0	9	14
Leptospirosis	0	0	0	0	0	1	1	0	0	0	1	1
Malaria	0	0	0	0	0	1	0	0	1	1	1	2
Measles	0	1	0	0	3	7	0	0	0	0	3	8
Melioidosis	0	0	0	0	0	1	0	0	0	0	0	1
Meningococcal Infection	0	0	0	0	26	8	0	0	4	3	30	11
	2	1	0	0	0	0	1	0	0	0	3	1

Mumps	0	1	0	0	0	0	0	0	0	0	0	1
Pertussis	0	1	0	0	2	1	0	0	0	3	2	5
Pneumococcal Disease	15	6	0	0	6	4	0	0	0	0	21	10
Rotavirus	25	27	4	6	42	5	24	0	6	3	101	41
Rubella	0	0	0	0	3	1	0	0	0	1	3	2
Salmonella	20	30	3	5	83	55	10	16	24	24	140	130
Shigella	14	4	1	4	17	8	8	11	1	1	41	28
Syphilis	16	44	27	16	19	7	21	5	12	2	95	74
Tuberculosis	1	2	0	0	1	4	0	0	2	2	4	8
Typhoid	0	0	0	0	0	2	0	0	0	0	0	2
Yersiniosis	0	1	0	0	0	1	0	0	0	0	0	2
Total	355	321	56	57	596	415	130	130	187	148	1324	1071

Points to note regarding notifications:

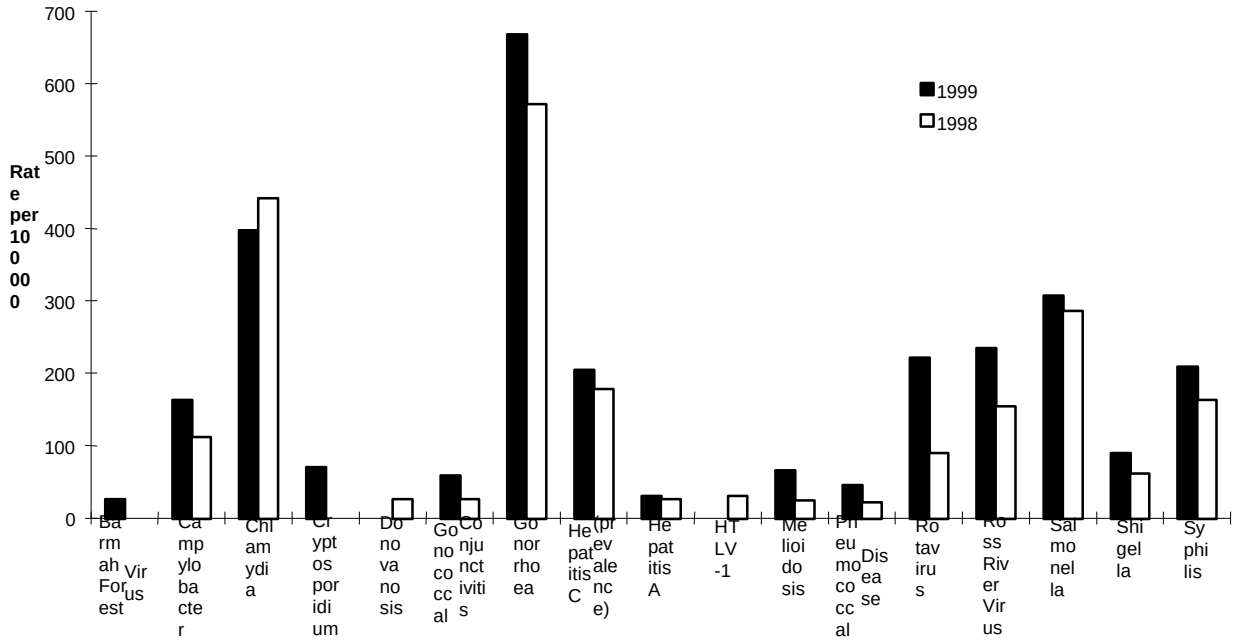
- Amoebiasis, Australian Encephalitis (MVE, Kunjin, Kokobera), Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Diphtheria, Gastroenteritis, Hepatitis C (incidence), Hepatitis D & E, Hydatid Disease, Leprosy, Listeriosis, Lymphogranuloma venereum, Poliomyelitis, Typhus, and Viral Haemorrhagic Fever are all notifiable but had "0" notifications in this period.
- The marked increase in melioidosis cases in the 1999 period reflect the late “wet” season and near record high rainfalls.
- The increase in gonococcal conjunctivitis cases in the Katherine district in the 1999 period was the result of an outbreak in January/February involving mainly two communities
- The decrease in donovanosis notifications this period was due to loss of a dedicated project worker (August 1998) and possible changes in diagnostic techniques.

NOTIFIED CASES OF VACCINE PREVENTABLE DISEASES IN THE NT BY REPORT DATE 1 JANUARY TO 31 MARCH 1999 AND 1998

DISEASES	TOTAL		No. cases among children aged 0-5 years	
	'99	'98	'99	'98
Congenital rubella syndrome	0	0	0	0
Diphtheria	0	0	0	0
Haemophilus influenzae type b	2	0	2	0
Hepatitis B	7	4	0	0
Measles	0	1	0	0
Mumps	0	1	0	1
Pertussis	2	5	1	0
Poliomyelitis, paralytic	0	0	0	0
Rubella	3	2	0	0
Tetanus	0	0	0	0

- Mumps is largely under-reported.

NT WIDE NOTIFIABLE DISEASES 1 JANUARY TO 31 MARCH 1999 AND 1998



Rates <10/100 000 not listed
NT est.resid.pop - 181 923 supplied by Epidemiology & Statistical Branch, THS

Correction to 1998 and 1997 NT Notifiable Diseases by District for Tuberculosis in last edition

DISEASE	ALICE SPRINGS		BARKLY		DARWIN		EAST ARNHEM		KATHERINE		TOTAL	
	'98	**'97**	**'98**	**'97**	**'98**	**'97**	**'98**	**'97**	**'98**	**'97**	**'98**	**'97**
Tuberculosis	2	4	1	0	20	24	0	4	9	4	32	36

STAFF UPDATES

CDC staff farewelled **Angela Merianos** on 11 June 1999. Angela came to CDC Darwin in 1991 to do the Masters of Applied Epidemiology through the National Centre for Epidemiology and Population Health (NCEPH). After successfully completing the course, she joined the teaching team at NCEPH in Canberra for a year and then returned to Darwin in late 1993 to take up the position as Head of Immunisation and Surveillance, CDC. Angela plans to explore life on the East coast via a three month stop over in Dubbo.

A warm welcome to **Frank Bowden** back in Darwin from a year's study at the Wellcome Trust Centre for Epidemiology of Infectious Diseases, Oxford University, UK. Most will remember Frank as the Head of STD/AIDS from 1992-97. Frank will be filling Angela's former position as A/Head of Surveillance for the next three months.