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Acute Post Streptococcal Glomerulonephritis in the Northern Territory 1980 – 2000

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Introduction

Acute post streptococcal glomerulonephritis (APSGN), an inflammatory kidney disease that follows streptococcal skin or throat infection, remains a serious cause of morbidity in remote Aboriginal communities in the Top End of the Northern Territory (NT). APSGN outbreaks recur at regular intervals signalling circulation of nephritogenic streptococcal strains. Control of outbreaks requires intensive community interventions that aim to protect those most vulnerable to disease by reducing carriage of the bacteria in the community and opportunities for infection to occur.

Considered at the time a benign disease without long-term sequelae, the first well documented outbreak of APSGN in the NT occurred in early 1980 across several Top End communities. Later outbreaks occurred in 1983/1984, 1987, 1993, 1994, and 1995. In 2000 a large outbreak of ASPGN occurred across several Top End communities, 6 to 7 years following the last major outbreaks of disease. This paper summarises the history of ASPGN in the NT in the 20 years preceding the most recent outbreak. The aim of the paper is to add this information to the local health record.

Methods

Review of the Centre for Disease Control (CDC) records for APSGN. Available records for

Contents

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APSGN in the NT prior to 1991 consist of a paper based file that contains records of outbreaks and interventions, reports of district medical officers (DMOs) and case numbers for outbreak years. The documents within the file were collated and the information summarised.

From 1991 onwards, data about APSGN cases have been maintained as part of the NT notifiable diseases dataset. These data were examined from 1992-1999. Reports of NT disease outbreaks and NT Communicable Disease Bulletin articles were also searched.

History of APSGN in the NT 1980 – 1999

Outbreaks of APSGN have occurred in the NT since at least 1965, with outbreaks also reported as occurring in 1969 and 1973,[1] the latter being described in one correspondence as extensive. While there is no documentation available to describe these outbreaks they appear to follow a similar pattern of intermittent occurrence as those reported in later years. Although outbreaks of APSGN had been recognised, neither the 1973 'Bush Book'[2] or the subsequent version published in 1979[3] contained information about the disease, diagnosis or management. The stimulus for the development of a policy statement for the control of 'epidemic glomerulonephritis' was the outbreak of disease that occurred across several Top End communities in 1980.

1980. The first well documented outbreak of APSGN, called glomerulonephritis at the time, occurred in April and May 1980 with several hundred cases reported in communities across the Top End.[4] Cases were reported at Port Keats, the Tiwi Is. (Nguiu, Milikapiti and Pularumpi), Daly River, Borroloola and one other Top End community with details available for Port Keats and the Tiwi communities. Information about case numbers, the case definition used and interventions in response to the outbreak were initially documented in the form of reports by DMO's responsible for providing medical care to the communities.

The Port Keats outbreak commenced in the second week of April 1980 with 3 cases of clinical disease diagnosed within 3 days from 14 April 1980 and 72 cases by day 7. All cases had facial oedema and 69 had haematuria and proteinuria. Twelve were evacuated to Royal Darwin Hospital and the remaining 60 were treated in the local community health centre with fluid restriction, bed rest and medications as required. The community intervention consisted of screening all school children (n=250) for signs of the disease and administering prophylactic long acting penicillin (LA bicillin). Two months later 30 children randomly screened had persisting haematuria but were otherwise well.[5]

The Tiwi outbreak commenced on 23 April 1980 when 2 patients presented with clinical disease. Two case definitions were employed during this outbreak. Clinical disease was defined as the presence of oedema and haematuria with or without hypertension. Subclinical disease was defined as the presence of haematuria on dipstick in otherwise asymptomatic individuals. A total of 55 clinical cases were reported across the 3 Tiwi communities.

The community intervention employed to control the outbreak consisted of administration of LA bicillin to all school aged children. Fifty-one (92.7%) clinical cases were aged less than 15 years. Overall, 450 school children were screened and 158 (35.1%) were found to have haematuria (sub-clinical disease).

Forty-seven of the 55 Tiwi cases (85.5%) occurred at Nguiu, with the outbreak there lasting approximately 2 weeks. Forty-one clinical cases occurred at Nguiu in a total school aged population of 356 (ages 4 to 17 years) giving an attack rate for school children of 11.5%.[6]

The documentation of other outbreaks that year were more limited with the subclinical disease (haematuria) reported more commonly than clinical disease. Eighteen cases of 'haematuria' were reported at Borroloola in June and July 1980. By August, development of a policy for prevention and early detection, outbreak control and clinical management of APSGN had commenced. The policy recommended that all children and adults with haematuria and skin sores be treated with a stat dose of LA bicillin. People with haematuria and oedema were to be reported to the DMO by radio.[1]

Although communities in West Arnhem appear to have escaped an outbreak, cases further east were reported. Four children were admitted to Gove District Hospital in August with APSGN, 2 each from Numbulwar and Angurugu and in September, 66 children from Numbulwar were found to have haematuria on school screening. Eight cases of 'mild' disease (which is undefined),

occurred over 4 months in Gove and Yirrkala (reported September) and this was noted to be a greater than normal prevalence of disease. Forty otherwise asymptomatic children had haematuria at school screening at Galiwin'ku in September.

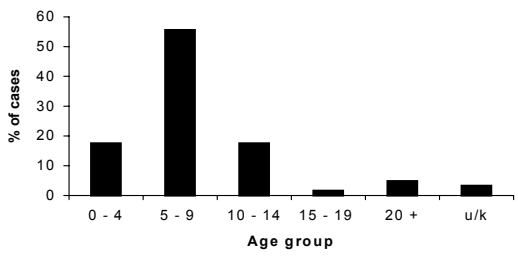
Few cases of APSGN were reported in the Alice and Barkly regions, however a September letter of the regional manager at the time revealed that there had been 6 paediatric APSGN admissions to the Alice Springs Hospital in the previous 6 months with this the normal prevalence of disease.

1983/1984. An outbreak involving Groote Eylandt and Numbulwar in December 1983 to January 1984 was referred to in a 1987 report. There is no information about this outbreak contained in the file.

1987. A major outbreak of APSGN occurred between May and July 1987, originating in East Arnhem and affecting 7 communities.[7] Sixty-three cases of disease were reported, however it is uncertain what case definition for disease was used as none are referred to in correspondence. Hospital admission of 32 cases (50.1%) suggests that this number at least had clinical disease and community treatment of the remaining 31 is mentioned though details are not recorded. More males (n=34, 54%) than females (n=29, 46%) had disease. Fifty-seven cases (90.5%) were younger than 15 years and case ages ranged from 2.5 to 42 years. Children aged 5 – 14 years were treated prophylactically with LA bicillin, however levels of coverage were not reported.

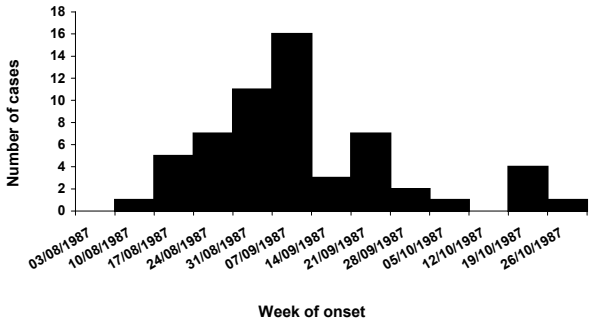
There are no data available about the dates of onset across the individual communities. Seven cases occurred at Groote Eylandt, 13 at Ramingining, 3 at Gapuwyiak, 16 at Milingimbi, 20 at Galiwin'ku, 3 at Nhulunbuy and a single case at Numbulwar.

Figure 1 Age of cases. APSGN outbreak, East Arnhem, NT. 1987



In August 1987, an outbreak of APSGN lasting 12 weeks commenced on Bathurst Island. Fifty-eight cases of clinical disease (facial oedema, weight gain, haematuria, proteinuria and hypertension) occurred with laboratory results confirming diagnosis for all cases. Fifty-five (94.8%) cases were less than 15 years of age, 32 (55.2%) were male and 26 (44.8%) female.[8]

Figure 2 Epi-curve. APSGN outbreak Bathurst Island, NT. 1987



The initial prophylaxis protocol for this outbreak was to treat the siblings of child cases with LA bicillin. In week 5 of the outbreak, the treatment protocol was changed and all children less than 12 years were given LA bicillin.[8]

APSGN notifications 1991 – 1999

In 1991, electronic storage of disease notification data on an Epi-Info database commenced and yearly APSGN data were maintained for all recorded cases and outbreaks of disease.

1993. Six years following the 1987 outbreak, 25 cases of APSGN occurred in East Arnhem in 4 communities. The cases occurred over a 4 month period at Milingimbi (n=12), Galiwin'ku (n=5), Gapuwiak (n=3) and Yirrkala (n=5). The age range of these cases differed from those reported in previous outbreaks with 15 (62.5%) aged less than 15 years and 18 (75%) less than 20 years.

Table 1 Distribution, number and month of onset, APSGN cases, East Arnhem 1993

	8/93	9/93	10/93	11/93	Total
Galiwin'ku	1	2	2	0	5
Gapuwiak	0	2	1	0	3
Milingimbi	0	0	3	9	12
Yirrkala	2	3	0	0	5
Total	3	6	6	9	25

1994. 21 cases of APSGN were reported in 1994 with the only outbreak of disease occurring on Bathurst Island at Nguiu. There were 5 confirmed and 4 possible cases of disease in this outbreak (CDC records). The cases occurred over a 2 month period and 8 (88.9%) were children aged less than 15 years. There was no community intervention for this outbreak.

1995. This year saw another major outbreak of APSGN that affected communities to the east and west of Darwin. A total of 67 cases were reported for the year. Unlike earlier years, 11 cases were reported from southern NT including 5 cases in Ali Curung, a Barkly region community.

The same communities affected in 1993 experienced APSGN in 1995, the 2 year interval between disease occurrence differing from the previous pattern where the interval was longer. There were 25 cases in 4 communities over 4 months, however only one community, Gapuwiak experienced a recognised community outbreak of disease. The overall East Arnhem case age range was 2 to 50 years, 70.8% (17/24) were less than 15 years and 83.3% (20/24) were less than 20 years. Milingimbi, which had the most cases in 1993 (n=12), had the least in 1995 (n=2).

Table 2 Distribution, number and month of onset. APSGN cases, East Arnhem 1995

	4/95	5/95	6/95	7/95	8/95	Total
Galiwin'ku	0	1	4	0	1	6
Gapuwiak	0	9	2	1	0	13
Milingimbi	0	1	0	1	0	2
Yirrkala	0	0	2	2	0	4
Total	0	11	8	4	1	25

Two other communities had outbreaks of APSGN in 1995, Port Keats and Ngukurr. The 1995 outbreak was the first reported in Port Keats since 1980 and the first ever reported for Ngukurr.[9] There were 10 cases of disease notified from Port Keats and unlike those reported in the east that year all were children less than 15 years (age range 3 to 10 years). In Ngukurr, 4 cases of disease were reported and 5 subclinical cases were identified. The case age range was 10 to 16 years.[9] In both Ngukurr and Port Keats, the community interventions consisted of prophylactic treatment of all community children aged 3-15 years, all household contacts aged 0 to 15 and all community children aged less than 3 with skin sores or sore throat with LA bicillin.

APSGN cases 1991 – 1999

In the past decade specifically between 1991 and 1999, 177 cases of APSGN were reported to CDC. Age was reported for 173 (97.7%) cases. One hundred and forty-six cases (84.4%) were children aged less than 15 years and 91.3% were aged less than 20 years. The first notification from a southern NT community was in 1994 at a small eastern Central Australian community and cases were reported in the Barkly and Central Australia in 1995. Intermittent notifications of disease from these regions have continued generally as single cases reports.

Figure 3 Yearly ASPGN notifications in the NT 1991 – 1999

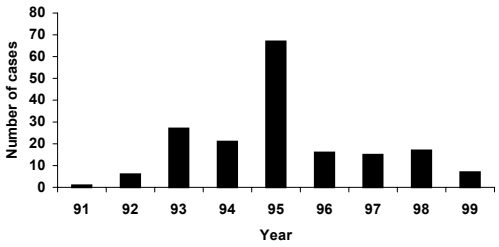
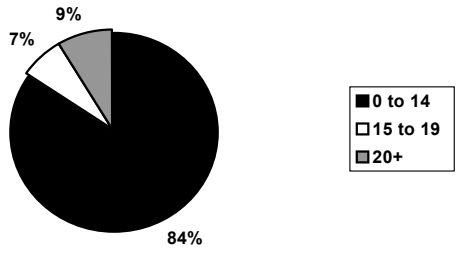


Figure 4 NT APSGN cases by age 1991 – 1999



Discussion

Outbreaks of APSGN have occurred at irregular intervals in remote NT Top End communities since the 1960's. The 1980 outbreaks of ASPGN were explosive, with rapid rises in case numbers in the main communities affected i.e. Port Keats and Nguiu. Prophylactic programs developed in

response to the outbreaks were directed primarily at school aged children and in both communities the outbreaks subsided relatively quickly. Whether the rapid decline in case numbers resulted from the prophylaxis programs or spontaneously, as most or perhaps all susceptible children had already been exposed and developed disease, is unknown. The explosive nature of the outbreaks may indicate the latter.

The outbreaks that occurred in 1987 were more protracted than those earlier, however relatively large numbers of cases were still being reported. In the 1990's the outbreaks of disease were smaller than those reported previously. Whether this resulted from a change in the interventions used during outbreaks or the interplay of factors that influence transmission and carriage of the organism, including prevalence of skin sores and scabies and or changed living conditions is a matter for speculation.

Interestingly, many of the NT outbreaks of APSGN have occurred across groups of communities where there is likely to have been travel links to facilitate spread of the streptococcal strains responsible for disease. In the 1980 outbreak, the communities in Darwin Rural District where outbreaks occurred, had either language group or administrative ties and so travel links. Outbreaks in East Arnhem regularly recur in those communities that have language group and travel ties. Knowledge of these links and the epidemiology of past outbreaks assists rapid responses and pre-emptive disease alerts that may result in timely outbreak interventions and best case management.

The first NT guidelines for the control of disease were developed in 1980 in response to the widespread outbreaks of APSGN across the Top End. In many respects the guidelines were similar to those employed today. School aged children were the focus for interventions and adults with either skin sores or haematuria were also targeted for prophylactic LA bicillin. Unlike contemporary guidelines, case contacts were not specifically targeted and haematuria without other symptoms of disease was treated. There were 2 case definitions employed during earlier outbreaks; one described clinical disease (oedema, haematuria +/- hypertension) and the other, haematuria alone with no other symptoms. Irrespective of the definition used, all individuals who met either case definition received penicillin. The early guidelines also refer to the interventions as a method for interrupting the outbreaks of disease and describe the screening protocol to be employed.[1]

In 1987, different interventions were employed for the East Arnhem and Nguiu outbreaks. In the former, children aged 5 to 14 years were given prophylactic LA bicillin, although there are no records of coverage rates or when the intervention commenced. At Nguiu sibling contacts of cases were originally targeted in a prevention program, with the intervention later expanded to target all children less than 12 years.[8]

Following the widespread outbreak of disease in 1995 that affected communities to the east and west of Darwin, new guidelines[10] were developed by Darwin CDC staff and published in August 1997. Based on evaluation of local interventions and review of published observational data,[11] the guidelines define the disease, provide a community outbreak definition and describe the interventions to be employed in response to sporadic cases and community outbreaks. The contemporary case definition has both laboratory and clinical components and reflects the change in knowledge about the disease since the earlier outbreaks occurred. The role of penicillin in containing outbreaks of disease remains 'untested' as randomised control trials to assess its efficacy in outbreak situations have never been conducted. Interestingly, the worldwide lack of data about the effectiveness of administering penicillin for control of APSGN was recognised in 1980 and the situation remains unchanged in the intervening 20 years.

This manuscript was circulated to all APSGN involved communities and was endorsed for publication with minor revisions.

References

1. Northern Territory Department of Health. Policy Statement on Epidemic Glomerulonephritis Surveillance and Control. 1980.
2. Northern Territory Medical Services, Darwin. Bush Book. 1973.
3. Jacobs DS. ed. Northern Territory Bush Book. Northern Territory Department of Health. 1979.
4. Gogna NK, Nossar V, Walker AC. Epidemic of acute post streptococcal glomerulonephritis in Aboriginal communities. *Med J Aus* 1983; 1:64-66.

5. Rebgetz P. Glomerulonephritis outbreak at Port Keats. Northern Territory Department of Health. DMO Report. April 1980.
6. Rebgetz P. Acute glomerulonephritis. Northern Territory Department of Health. DMO Report. July 1980.
7. Madsen HD. Glomerulonephritis outbreak – East Arnhem region. Department of Health and Community Services. Report. July 1987.
8. Devanesen D. An outbreak of glomerulonephritis on Bathurst Island. Department of Health and Community Services. Draft report. December 1987.
9. Wallace T. Acute Post Streptococcal Glomerulonephritis (APSGN) – Outbreak at Ngukurr. *Comm Dis Bull* 1995; 2(6):1-3.
10. Centre for Disease Control, Territory Health Services. Guidelines for the Control of Acute Post-Streptococcal Glomerulonephritis. 1997.
11. Johnston, F, Carapetis J, Patel MS, Wallace T, Spillane P. Evaluating the use of penicillin to control outbreaks of acute post streptococcal glomerulonephritis". *Paediatr Infect Dis J* 1999; 18:327-32.

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Outbreak of Acute Post Streptococcal Glomerulonephritis in the Northern Territory - 2000

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Abstract

Objective: To describe the Acute Post Streptococcal Glomerulonephritis (APSGN) cases that were notified to Darwin Centre for Disease Control (CDC) in the Northern Territory (NT) from February to July 2000 and report on the outcomes of 6 community interventions that used benzathine penicillin and 5% topical permethrin to control the outbreak.

Methods: In the NT APSGN is notifiable. A database was established to record the laboratory findings and clinical features of the disease for each notification received from the Districts' Centre for Disease Control (CDC) or remote health teams. The case definition used was adapted from the Territory Health Services 1997 Guidelines for the Control of APSGN.

An outbreak was defined as 2 clinical cases in 1 week or 3 in 1 month. To control the outbreaks an intervention was implemented that involved screening and treating all children in the community between the ages of 0-15 years. Screening entailed inspecting each child for skin sores, scabies and oedema. If present, skin sores were treated with an injection of benzathine penicillin and scabies with 5% topical permethrin. If oedema was evident the child was investigated further for manifestations of APSGN. Data was collected on the screening and treatment administered for each child in the interventions that were implemented in 5 remote Aboriginal communities.

Results: Fifty-three cases of APSGN were reported between February to July 2000, all occurring in Aboriginal people. The median age was 9 years from a range of 1-39 years. Females were only slightly more represented than males 1.3:1. Of the 53 cases 46 (87%) had skin sores and 21 (40%) scabies. Haematuria was the most common symptom of disease in 52 (98%) of the cases, followed by oedema in 45 (85%) and hypertension in 37 (79%).

Twenty-six cases occurred in 5 communities that met the outbreak definition and implemented 6 community interventions. Almost two thirds (n=1426) of children aged 0-15 years in the 5 communities were screened for the presence of skin sores, scabies and oedema. Of the 1426 children screened, 860 (60%) had skin sores and 837 (59%) were treated with benzathine penicillin. There were 350 (24.5%) with scabies, and 325/893 (36%) were recorded as receiving 5% topical permethrin.

Conclusion: The risk associated with skin sores and scabies and APSGN compared to those children with neither skin ailments supports the continuation of both treatments during an outbreak. Preventive measures combined with prompt treatment to control associated skin conditions in the community would be the major contributors to hindering future outbreaks of APSGN.

Introduction

Acute post-streptococcal glomerulonephritis (APSGN) is an immune complex mediated response[1,2] that follows a Group A streptococcal (GAS) infection of the skin or pharynx.[3,4,5,6] The sequelae of streptococcal infections are uncommon in industrialised countries[7] but remain a problem in developing countries and in Indigenous populations.[8,9,10] APSGN has been reported in tropical[8], temperate[11] and arctic[12] climates in most months of the year and in many different cultural groups eg. Eskimos, Australian Aboriginals, Native American Indians. The epidemiology of outbreaks of APSGN in the Northern Territory (NT) has remained unchanged over the years, predominantly affecting the same age group in the same locations in the dry season months.[13]

APSGN most commonly affects children aged between 6-10 years but disease can occur in people of any age group.[2] Multiple strains of GAS are known to circulate at any one time[14,15] with only the nephrogenic strains causing disease[11,16,17] in non-immune individuals. Complete recovery is expected in 95%[18,19] of cases following the illness however there is a fatality rate of 1%[10, 19] and 1-5% of cases can progress to chronic glomerulonephritis.[18, 20] There is also now evidence to suggest that frequent mild episodes of APSGN [21] contribute to end-stage renal disease[22] in Australian Aboriginals.[17,23,24,25]

Sporadic cases of APSGN are reported each year in the NT[13] with large outbreaks (>50 cases) of the disease in the past 40 years occurring approximately every 5-7 years.[26] Due to the recurring outbreaks Territory Health Service (THS) introduced *Guidelines for the Control of APSGN*[3] in 1997 that were updated from the 1980 protocol. The strategy to only use benzathine penicillin to treat children aged 0-15yrs with skin sores is a major change from previous years in which entire communities were given penicillin regardless of skin sore status. The 1997 guidelines were first used in an outbreak in February 2000.

This paper reports on the manifestations of the APSGN cases and the results of the community interventions that screened children for skin sores and scabies and gave treatment accordingly. The epidemic curve for each community is discussed in relation to the epidemiology of the outbreak.

Setting

The Top End of the NT has a tropical climate with many of the related skin conditions. Over one quarter of the NT population are Aboriginal[27] and the majority live in remote locations. The communities that had identified outbreaks had populations that varied from 300 –1800 people,[28] and were all coastal besides one that was located on a lake. The communities were all, to some degree surrounded by mangroves, mud flats and salt bush where biting midges and mosquitoes are often encountered along with flies.[29,30,31,32,33] Insects are often implicated as a vector in the causation of sores or abrasions that become infected with GAS.[19, 34]

The prevalence of skin sores and scabies in remote Aboriginal communities have been known to be as high as 50% which enables the streptococcus to continue circulating in communities.[35] Streptococci can be found on normal skin for an average of 10 days before the development of skin lesions and the subsequent spread of infection is by direct contact with other infected individuals.[19] GAS have been documented to live in dust for months[36] and therefore major preventative measures include washing and hygiene.[19]

Methods

Notification of APSGN Cases

Clinical cases were notified by telephone or facsimile from the remote health teams or Centre for Disease Control (CDC) managers in outlying districts to Darwin CDC. The clinical case definition included 2 or more of the following manifestations: microscopic haematuria with 2+ or more of blood on urine dipstick, oedema and hypertension (diastolic blood pressure >90 if older than 13 years or >80 if <13 years).

Health practitioners were encouraged to support the clinical diagnosis with laboratory evidence of streptococcal infection and immune response according to the 1997 THS APSGN Control Guidelines which included:

1) Evidence of recent streptococcal infection by a positive group A Streptococcal skin or throat culture, or elevated serum antistreptolysis 0 titre or antiDNase B titre.
2) Reduced serum complement (C3) level.[5]

Benzathine penicillin was to be administered to all close and household contacts of cases aged 3-15 years, as well as contacts with skin sores regardless of age. If scabies were detected the entire family was to be treated with topical 5% permethrin. It was recommended that all contacts be screened for clinical manifestations of APSGN.

Community Outbreak definition

A community outbreak was defined as 2 clinical cases of APGSN in 1 week or 3 clinical cases in 1 month.[3] When the criteria for an outbreak was met it was recommended the community implement an intervention using the THS 1997 APSGN Control Guidelines.[3] A community intervention involved educating the community members about the disease and screening all children aged 0-15 years for skin sores and scabies.[3] Oedema was used as an extra screening tool to identify children with possible APSGN - this was not included in the 1997 APSGN Control Guidelines. Epidemiologically linked cases of APSGN were expected up to 6-8 weeks after an intervention.

A skin sore was defined as an open lesion on the skin. Scabies were identified as small papules and scratch marks round web spaces, on elbows, armpits, trunk, between fingers and toes and around wrists.[35] Pustules were also looked for on the head, palms and feet of babies.[35] Sores and scabies were identified by the health workers responsible for implementing the intervention. Benzathine penicillin was the recommended treatment for skin sores and topical 5% permethrin for scabies.[3] Any child identified with oedema was investigated further for APSGN.

Population lists were not obtained from the same source for all the communities. Three of the interventions used population lists collated by the THS Epidemiology branch, one used the District Medical Officers database and the other community used their own population list maintained by the clinic. The population lists were used to identify the number of 0-15 year olds that required screening and to record the outcome of the screen and the treatment administered. These lists were then forwarded to a central person at CDC Darwin for collation and analysis.

Statistical analysis

Data was entered in Stata 5[37] and analysed in Stata 5 and Epi Info Version 6.1.[38] The risk ratios (RR) were calculated using the data from the children screened and the cases from those communities that were aged 0-15 years. The 95% confidence intervals (CI) for the RR were calculated assuming a binomial distribution.

The attack rates were calculated for the 0-15 year population using correct ages at the time of the intervention. Immune status and exposure were unknown for most individuals screened however the attack rate was calculated using the number of cases in the 0-15 year population divided by the total population of children aged 0-15 years.

Results

APSGN Cases

There were 53 cases of APSGN notified from 15 different remote Aboriginal communities during February-July 2000. Of the 53, 9 were sporadic cases occurring in communities that did not meet the outbreak definition and 14 of the 53 cases were family relatives or living in the same house. The median age was 9 years ranging from 1-39 years. Thirty (56.6%) were female and 23 (43.4%) male, all Aboriginal. Of the 53 cases 21 (40%) had scabies and 47 (87%) had skin sores.

Of those that presented with a clinically compatible illness 52 (98%) had haematuria, 45 (85%) had oedema and 37 (70%) had hypertension. In total 36 (68%) cases had culture swabs taken of which 11 were throat swabs and 25 skin swabs. GAS was only isolated from 14 (26%) cases, all of whom had skin sores. M3 was the only nephritogenic type of circulating GAS identified from the cases. An elevated antistreptolysis and AntiDNase B titre was present in 11 (20%) and 42 (79%) respectively of cases with 46 (87%) having evidence of a reduced serum complement 3 (C3) level.

Two thirds of the cases required hospitalisation for management of their hypertension and the other third were monitored in the community setting. There was 1 fatality, a female aged 13 years but the definitive cause of death is still with the coroner. Benzathine penicillin and 5% permethrin were administered to close and household contacts with skin sores and/or scabies, however separate details on these individuals was not collected for all the communities. No close or family contacts were identified with clinical or subclinical APSGN when screened.

Community Intervention

Of the 15 communities that notified APSGN cases, 7 communities met the outbreak definition and implemented interventions. Of the 7 communities, 2 implemented 2 interventions each for 1 indication of an outbreak and a third implemented an intervention each for 2 separately identified outbreaks making a total of 10 interventions. Staff numbers implementing the interventions varied from 2-8 people depending on the size of the community and availability of health related workers. Screening and treatment was implemented in the clinic, schools, community and people's homes. The outcomes of each child seen were recorded on the population lists.

Twenty-six cases reported were from 5 of the communities that implemented 6 interventions that will be discussed in this paper. Almost two thirds (n= 1426, 61%) of the target population of children aged 0-15 years were screened. The proportion of children screened in each community ranged from 46-100% of whom 860 (60%) had skin sores and 350 (24.5%) had scabies.

In 5 (n=1069) of the interventions skin sores were most common (25.5% n=273) in the 5-9 year age group and were approximately 1.5 times greater in this age group compared to those 0-4 and 10-15 years. However the percentage of scabies was only marginally higher (7.1% n=76) in the 0-4 year age group compared to the 5-9 (6.7% n=72) and 10-15 (5.7% n=61) year age groups.

The children with skin sores were 5 times more at risk of acquiring APSGN than those with no sores (RR5.74 CI 1.75, 18.84) whereas the children with scabies had twice the risk of acquiring ASPGN compared to those with no scabies (RR2.30 CI 1.13, 4.68).

Treatment with benzathine penicillin (n=837) was generally reflective of the prevalence of skin sores however scabies treatment was recorded in only 4 of the 6 interventions with 325 (36%) receiving 5% topical permethrin (Table 2). Only 1 person during the screening process was identified with oedema and was subsequently diagnosed with APSGN.

The crude attack rate for each community ranged from 0.3-4.6% (Table 1).

Table 1 Attack rate for children aged 0-15 years in the communities that implemented interventions during Feb-June 2000

	Population 0-15 years	No. of cases 0-15yrs	Crude Attack Rate (%)
Milingimbi	351	8	2.2
Ramingining	294	1	0.3
Maningrida	833	5	0.6
Croker Is	65	3	4.6
Nguiu 1	384	4	1.0
Nguiu 2	413	9	2.1

Table 2 Results of the skin status and treatment given to children aged 0-15yrs who were screened in the community interventions during Feb-June 2000

	% screened	% skin sores	% LAB	% scabies	% lyclear
Milingimbi	56	54	53	8	15
Ramingining	59	43	45	20	17
Maningrida	54	61	58	16	55
Croker Island	100	51	51	23	31
Nguiu 1	93	63	61	40	NR
Nguiu 2	46	78	76	36	NR

Epidemiology of the community outbreaks

Nguiu had 3 clusters of cases in Feb, May and July with a total of 13 cases reported, all aged between 3-10 years (Figure 1). All of the cases had skin sores and eight had scabies. Interventions conducted in February and June screened 357 (93%) and 190 (45%) respectively of the targeted population. No-one in the first cluster of cases was related however in the 2 further subsequent clusters, 2 of the 5 and 3 of the 4 cases were household family relations with the fourth case in the third cluster being a family relation to a case in the second cluster. The third cluster of cases fell within 8 weeks of the second cluster of cases so a joint decision between the community and health staff was made not to implement another intervention but to extensively screen and treat all household and other close contacts of the cases who had skin sores and scabies.

Milingimbi had 11 cases aged 1-16 years over a 7 week period and implemented 2 community interventions over a 3 week period (Figure 2). Of the 11 cases, 10 had skin sores and 4 had scabies. The combined interventions screened 211(60%) of 351 children. The first intervention screened 21% & the second a further 39% of the targeted population. The cases came from three different camps, 2 were living in the same house and another 2 were related but living in different houses. Of the 2 cases living in different houses the case onset dates were 3 weeks apart. The second case for that family was a child of aged 1 year who did not have skin sores and thus did not receive benzathine penicillin as a contact. From local knowledge it was confirmed that the children within the 3 different camps all lived close together and played with one another.

Croker Island reported 3 cases aged 7-10 years that were all notified on the same day from 3 different households (Figure 3). One case had both skin sores and scabies and the other 2 had neither. The children all lived close to one another and played together. The community intervention lasted 4 days and was completed 9 days after the cases had been notified. At the time of the intervention 55 children in the target population were on island and all (100%) were screened.

Three unrelated clinical cases were notified in Ramingining in 1 week. The intervention was implemented over 4 days screening 171 (58%) of the 294 children aged 0-15 years. Case notes were reviewed at the time of the intervention and 1 of the cases notified did not meet the case definition; however another case was found after reviewing family contacts (Figure 4). Of these 3 cases, 1 had no skin sores or scabies, another had both skin sores and scabies and the third case had no scabies but skin sores. The age range of the 3 reported cases was 13-39 years. Two cases were from the same house, the child was diagnosed in the first week and his uncle a week later. Of these 2 cases 1 had been in Milingimbi when their cases were first diagnosed.

Maningrida had 5 cases aged 9-13 years notified over a 1 month period. Two of the children lived in the same house and the other cases were from different camps in the community (Figure 5). All the cases had skin sores but only 2 had scabies. During the 2 week intervention 453 (54%) of the 833 0-15 year olds were screened. At the same time staff were implementing a Healthy Skin Program that continued after the intervention period. Precise records for the Healthy Skin Program were not kept but many more children would have been screened and treated after the intervention that have not been recorded.

Galiwinku notified 3 cases aged 6-17 years in a 3 week period (Figure 6). All of the cases had skin sores and 1 had scabies. As the surrounding communities were experiencing outbreaks this community implemented an intervention after only 1 case was notified. The health staff opened the clinic on a Saturday and relied on the parents to bring their children to the clinic for screening where approximately 50 children were seen. The second intervention following the second and third cases reportedly went out to the houses in the community and to the school to screen the children but no data is available on numbers screened and treated.

There were 5 cases aged 2-16 years notified by Yirrkala in a month, none of whom were related. None of the cases had scabies but 2 had skin sores. The community intervention was implemented over a 2 day period. No data is available on the numbers screened or treated.

From the 7 communities that had outbreaks the shortest period a community was affected per outbreak was 2 weeks and the longest period was 13 weeks with an average of 6.2 weeks per outbreak. The average length of an intervention was 6.6 days from a range of 3-9 days. The staff available for each intervention varied from 2-8 people.

The graphs illustrate the number of cases notified for each community. The 0 on the x axis represents when the community intervention was implemented and shows the weeks preceding and following the interventions. The black bars show cases that are unrelated and the white and stripe bars illustrate familial or household cases.

Figure 1 Nguiu APSGN cases by week of onset Feb-July 2000

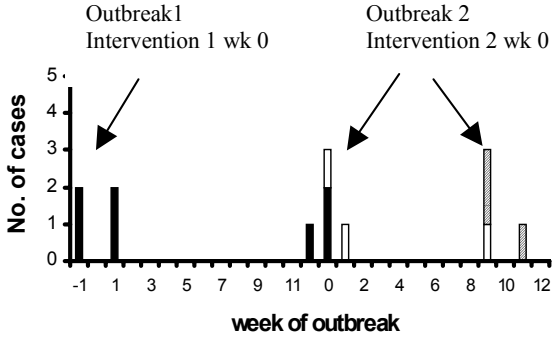


Figure 2 Milingimbi APSGN cases by week of onset April-May 2000

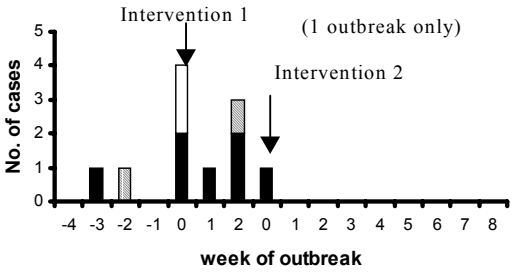


Figure 3 Croker Island APSGN cases by week of onset April 2000

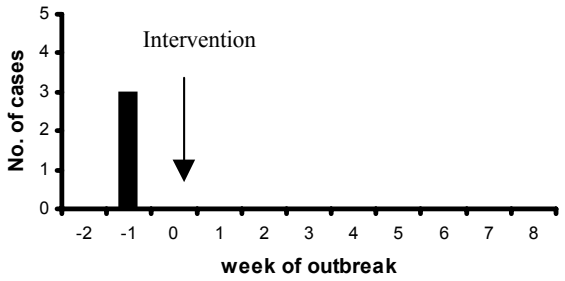


Figure 4 Ramingining APSGN cases by week of onset April-May 2000

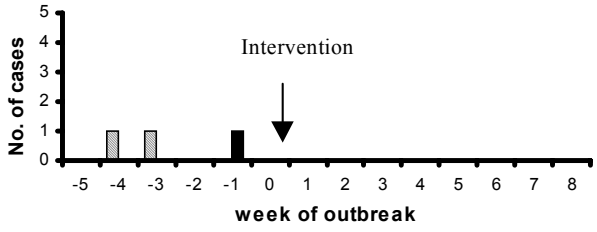


Figure 5 Maningrida APSGN cases by week of onset May-June 2000

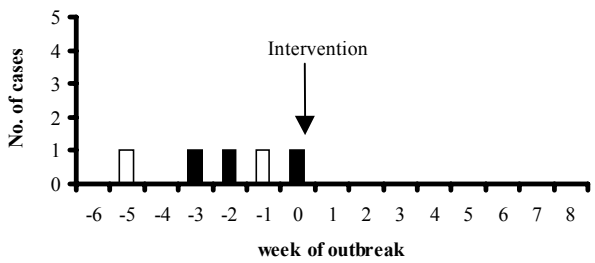


Figure 6 Galiwinku APSGN cases by week of onset May 2000

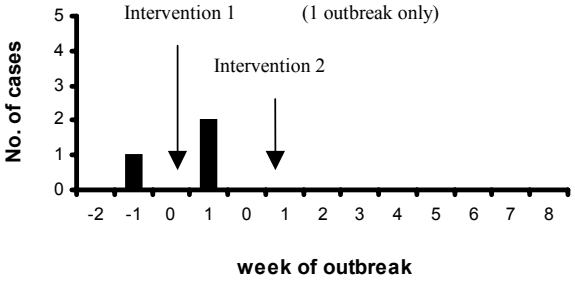
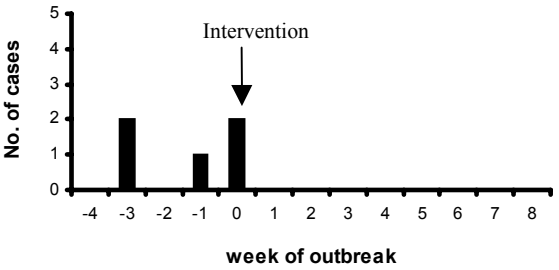


Figure 7 Yirrkala APSGN cases by week of onset May-June 2000



Discussion

Familial spread of GAS skin infections[7] and subsequently APSGN are common[19] as was seen with this outbreak in which over one quarter (26.5%) of the cases were related. Skin lesions infected with GAS are most common in infants and preschool children[19] aged less than 5 years[7] but APSGN cases are generally found in those aged between 5-10 years.[2, 39] During this outbreak the majority of skin sores were found in those aged 5-9 years which differs from what has been documented previously. However this most likely reflects the school age children that were an easier group to target when screening. Fifty five percent of the APSGN cases were in the expected age group and 98 % were 17 years or less.

Many of the APSGN cases were not swabbed for GAS before receiving benzathine penicillin. From the 36 (68%) APSGN cases that were swabbed, only 14 (39%) of these cultured GAS from skin sores. This is contrary to other outbreaks that have recovered GAS, mostly from skin lesions, in over 60% of APSGN cases.[9,12,40] Other bacteria and viruses that have been implicated in acute postinfectious glomerulonephritis are staphylococcus, pneumococcus, Coxsackie virus B_4, echovirus

type 9, influenza and mumps.[39] Staphylococcus was isolated in 13 (36%) of the 36 cases who were swabbed, however 7 (19%) of these cultured both streptococcus and staphylococcus.

Other indicators of APSGN infection were mostly consistent with previous outbreaks reported [9,15,41] except for the ASOT titres. A study in Queensland (QLD) found that 82%[9] of ASOT were elevated whereas only 20% were elevated in this outbreak. Elevated ASOT are most often seen after pharyngeal infections with generally only a slight or no increase observed after skin infections[6,19] and antibiotic use.[17] The ASOT results were obtained from 3 different laboratories in the NT however all used the manufacturers recommended reference range (Personal communication - Thomas B, Murray R & Pederick W). The ASOT results from the NT outbreak are therefore consistent with the documented literature.

Generally in APSGN epidemics only one specific M type is predominant.[19] For this outbreak M3 was identified as the nephritogenic strain from 3 of the cases (Personal communication A Develecchio). In previous outbreaks in the NT, M55 (1980)[39] and M1 (1987)[36] have been implicated. The 5-7 year cycle of large APSGN outbreaks in the NT follows similar trends to Trinidad in the Caribbean, that has reported 6 year cycles of epidemic disease caused by distinct strains of streptococcus.[35]

The prevalence of skin sores and scabies in NT communities have been reported as being as high as 50%.[35] During this outbreak skin sore prevalence was almost 3 times greater than scabies prevalence. Of the 6 interventions reported on, Ramingining, Maningrida and Croker Island used the same person to coordinate the intervention, Nguiu used the same team twice and Milingimbi used 2 different teams of staff over the 3 weeks. The differing staff used in conducting the interventions may have had an impact on the varying numbers of sores and scabies that were identified in each community.

Benzathine penicillin has been used in many APSGN outbreaks but to date there have been no randomised controlled trials to asses the effectiveness of penicillin in controlling outbreaks.[20] The evidence to support the use of benzathine penicillin in outbreaks is based on its effectiveness in eradicating GAS infection from communities.[42] Benzathine penicillin is long acting and can be detected in sera up to 4 weeks after an injection in adults (depending on the dose given),[43] but it has not been proven to avert the development of APSGN in people in the latent phase of the disease.[9] The use of penicillin to control outbreaks has increasingly narrowed. Previous NT outbreaks initially treated all community members, then all children and outbreak interventions now target all household contacts of clinical cases who are 3-15 years old, all other household contacts with skin sores and all children up to 15 years old in the community with skin sores. In 7 of the communities that used the more targeted approach of treating those with skin sores and scabies only 1 community had cases after the intervention.

In previous published articles on the use of penicillin in APSGN outbreaks the time frame that cases would be expected after an intervention has never been described in detail. From a review of the literature for this outbreak, 8 weeks was contrived as a cut off for when cases could be expected following an intervention and when a new outbreak would be defined. We came to this conclusion because APSGN can develop 1-4 weeks after a streptococcal infection[2] and benzathine penicillin is effective in eradicating streptococcal infection for up to 4 weeks after administration in adults[35]. While taking into account the fact that benzathine penicillin will not avert disease if it is in the latent phase of developing.[19]

Therefore theoretically, depending on the GAS being present and the timing of the administration of benzathine penicillin to a targeted group, cases of APSGN can be expected up to 6-8 weeks after the notification of a new case. However if an intervention was performed and all of the GAS eradicated from the community, cases would only be expected in the following 4 weeks.

For the 3 communities (Nguiu, Milingimbi and Galiwinku) that implemented more than 1 intervention all cases post intervention fell within the expected 8 week time frame. The reasons for repeating interventions in Milingimbi and Galiwinku was due to the perceived small number of people screened and treated in the first intervention. Nguiu's 2 outbreaks occurred over a 26 week period, 10 and 8 weeks apart and were considered to be 2 separate outbreaks. However there was a considerable difference in the amount of children screened and treated between the 2 interventions (93% in the first and 46% in the second).

In regards to interventions using penicillin, no studies have reported on the amount of children that need to be screened and treated to make an intervention successful. Excluding Nguiu, in all the interventions that screened 54% or more of the children, no further cases were reported. The third cluster cases in Nguiu that occurred after the second intervention have been interpreted as cases that fell within the 8-week time frame. However the epidemic curve could also be interpreted as an intervention failure as only 45% of children aged 0-15 years were screened which could be construed as not being adequate to control an outbreak.

Three communities had cases that were family relatives or living in the same house (Nguiu, Milingimbi & Ramingining). The cases in each subsequent Nguiu outbreak were more closely related to one another than in the previous outbreak. The epidemic curves for the 3 communities with familial cases show that all except 1 case in Nguiu occurred with 4 weeks of one anther. The child in the third cluster of Nguiu cases was related to those in the second cluster and had not received penicillin as a contact. His contact with the other familial cases in the third cluster is unknown.

An unpublished report from Menzies School of Health Research using molecular typing (Vir typing) was carried out on 56 specimens that were implicated in the outbreaks (Personal communication A. Delvecchio). The results showed that out of the 6 specimens that were APSGN cases there were 4 representative strains circulating during the 6 month period. These comprised of 3 separate strains representing 3 cases and 1 strain being clonal for the other 3 cases. One type was present in 3 communities over a 6 week period. Two individuals from 1 community with onset dates of illness 15 days apart were infected with 2 different strains. A child that was a case in February cultured the nephritogenic strain in July in a skin sore but showed no other signs of APSGN. The strain cultured was a clone of the strain that had been circulating in February 2000. Type specific antistreptococcal immunity is known to be protective[18] if the immune response has not been curtailed by effective antimicrobial therapy.[18, 19, 44]

In future outbreaks of APSGN in the NT more culture specimens from cases and non cases are needed for typing to ascertain the different strains of Group A streptococcus circulating at the time. Data collection including epidemiological links from all interventions is needed on cases, contacts and the community screened to compare with previous years to be able to determine the best regimen of penicillin use. The continuation of 'healthy skin' programs targeting scabies and skin sores can only be beneficial in reducing the port of entry and subsequent immune mediated response of APSGN.

Conclusion

The association between skin sores and scabies and the development of APSGN reinforces the need for prevention of both skin ailments to successfully avert future cases and subsequent outbreaks of this disease. The importance of locating and treating all household and close contacts is a priority in the prevention of further cases and to avoid the need for an intervention.

References

1. Avery M & First L. Pediatric Medicine. Second ed.Williams & Wilkins; 1994.
2. Cotran R, Kumar V& Collins T. Robbins Pathologic Basis of Disease. Six ed.WB Saunders Company; 1999.
3. Disease Control Program. Guidelines for the Control of Acute Post-Streptococcal Glomerulonephritis. 1997. Territory Health Services.
4. Johnston F. Acute Post-Streptococcal Glomerulonephritis. Summary of the outbreak and results of community screening at Wadeye 1995.
5. Kleinman H. Epidemic Acute Glomerulonehpritis at Red Lake. Minnesota Medicine 1954;37:479-83.
6. Nissenson A. Poststreptococcal Acute Glomerulonephritis: Fact and Controversy. Annuals of Internal Medicine 1979;91:76-86.
7. Carperis J, Currie B & Kaplan L. Epidemiology and Prevention of Group A Streptococcal Infections: Acute Respiratory Tract Infections, Skin Infections and their Sequelae at the Close of the Twentieth Century. Clinical Infectious Diseases 1999;28:205-10.
8. Nimmo F, Tinniswood D, Nurrall, Baker G & McDonald B. Group A streptococcal infection in an Aboriginal community. The Medical Journal of Australia 1992;157:521-2.
9. Streeton CL, Hanna J, Messer R & Merianos A. An epidemic of acute post-streptococcal glomerulonephritis among Aboriginal children. Journal of Paediatrics and Child Health 1995;31:245-8.
10. Territory Health Services. Children's Standard Treatment Manual. 1995.
11. Markowitz M. Streptococcal disease in developing countries. Pediatric Infectious Disease Journal 1991;10 (10):S11-S14.
12. Magnolis H, Lum M, Bender T, Elliott S, Fitz-

gerald M & Harpster A. Acute Glomerulonephritis and Streptococcal Skin Lesions in Eskimo Children. American Journal of Diseases in Children 1980;134:681-5.
13. Centralised Notifiable Disease Database. Acute Post-Streptococcal Glomerulonephritis. 1991-1999.
14. Carapetis J, Gardiner D, Currie B & Matthews J. Multiple Strains of Streptococcus pyogenes in Skin Sores of Aboriginal Australians. Journal of Clinical Microbiology 1995;33 (6):1471-2.
15. Meekin G & Martin D. Autumn - the season for post-streptococcal acute glomerulonephritis in New Zealand. New Zealand Medical Journal 1984 Apr 11;):226-32.
16. Dillon H. Post-streptococcal Glomerulonephritis Following Pyoderma. Reviews of Infectious Diseases 1979;1 (6):935-42.
17. Van Buyunder P, Gaggin J,Maring D, Pugsley D & Matthews J. Streptococcal infection and renal disease markers in Australian Aboriginal children. The Medical Journal of Australia 1992;156:537-40.
18. Feign R & Cherry J. Textbook of Pediatric Infectious Diseases. Fourth ed.WB Sanders Company; 1998.
19. Hoeprich P, Jordan M & Ronald A. Infectious Disease A treatise of Infectious Processes. Fifth ed.JB Lippincott Company; 1994.
20. Johnston F Carapetis J, Patel M, Wallis T & Spillane P. Evaluation of the use of penicillin to control outbreaks of acute post-streptococcal glomerulonephritis. Journal of Pediatric Infectious Diseases 1999;18 (327):332.
21. Carapertis J. Ending the heartache: the epidemiology and control of acute rheumatic heart disease in the Top End of the Northern Territory. (Thesis) 1998 Aug.
22. Baldwin D. Post-streptococcal Glomerulonephritis A Progressive Disease. The American Journal of Medicine 1977;62 (1):1-11.
23. Hoy W. Renal disease in Australian Aboriginals. Medical Journal of America 1996;165:126-7.
24. Goodfellow A, Hoy W, Sriprakash, Daly M, Reeves M & Matthews J. Proteinuria is associated with persistence of antibody to streptococcal M protein in Aboriginal Australians. Epidemiol Infect 1999;122:67-75.
25. Hoy W, Matthews J, McCredie D, Puglsely D, Hayhurst B, et al. The multidimensional nature of renal disease: Rates and associations of albuminuria in an Australian Aboriginal community. Kidney International 1998;54:1296-304.
26. Evans C. Acute Post Streptococcal Glomerulonephritis in the Northern Territory 1980 -2000. The Northern Territory Communicable Diseases Bulletin 2001.
27. Australian Bureau of Statistics. Population Distribution, Indigenous Australians. 1996
28. Territory Health Services. Doing it Better for the Bush 1997.
29. NT Government of Health and Community Services. Vector Mosquito Survey Milingimbi and Ramingining. 1983 Jun
30. Vector Mosquito Survey, Maningrida. 1984 Apr
31. Vector Mosquito Survey, Galiwinku. 1984 Apr
32. Aedes Aegypti and Vector Mosquito Survey, Nguiu. 1988
33. Territory Health Services. Biting Midges and Mosquitoes in the Darwin Area. Northern Territory Government; 1995 Jun
34. Gorbach S, Bartlett J & Blacklow N. Infectious diseases. Second ed.WB Saunders Company; 1998.
35. Disease Control Program. Guidelines for Community Control of Scabies and Skin Sores. 1997. Territory Health Services.
36. Relf W. The molecular biology of the M protein of Streptococcus Pyogenes. 1993 Mar.
37. Stata Statistics/Data Analysis. Version 5. 1998.
38. Epi Info, Version 6: a word processing, database, and statistics program for epidemiology on microcomputers. Centers for Disease Control and Prevention; 1994.
39. Rudolph A, Hoffman J & Rudolph C. Rudolph's Paediatrics. Twentieth ed. A Simon & Schuster Co.; 1996.
40. Dillon H, Reeves M & Maxted W. Acute Glomerulonephritis following skin infection due to Streptococci of M-Type 2. The Lancet 1968 (Mar 16;):543-5.
41. Gogna N, Nossar B & Walker A. Epidemic of acute poststreptococcal glomerulonephritis in Aboriginal communities. The Medical Journal of Australia 1983 (Jan 22;):64-6.
42. Ferrieri P, Dajani AS & Wannamaker L. Benzathine penicillin in the prophylaxis of streptococcal skin infections: A pilot study. The Journal of Paediatrics 1973 Oct: 572-7.
43. The Mims Annual. Australian Edition ed.Crows Nest; 1997.
44. Mandell G. Mandell, Douglas Bennett's Principles and Practices of Infectious Diseases. 5 ed. Churchill Livingstone; 2000.

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The Top End rheumatic heart disease control program I. Report on program objectives

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Abstract

Objectives: To describe the outcomes of the rheumatic heart disease (RHD) control program in the Top End of the Northern Territory (NT).

Methods: The objectives of this program are: to create and maintain a computerised register of people in the Top End with acute rheumatic fever (ARF) and RHD, to develop methods of improving adherence to secondary prophylaxis, to provide appropriate health education for people with ARF and RHD, to provide support to health staff working in remote areas, to standardise the clinical care of people in the Top End with ARF and RHD, and to provide prospective epidemiological data to monitor the incidence and prevalence of ARF and RHD in the Top End of the NT.

Results: An ARF and RHD register and recall system has been developed. The program has worked closely with Top End remote area communities to identify problems surrounding adherence with benzathine penicillin prophylaxis. ARF and RHD education packages have been developed and distributed. Health service provider education is conducted through Territory Health Services, Aboriginal Medical Services and Aboriginal health worker training facilities. ARF and RHD reports are generated for remote area health centres.

Conclusions and implications: The Top End RHD control program has achieved most of its initial objectives but measuring and improving adherence to secondary prophylaxis remains a challenge. The program will be more fully integrated into routine services in the coming years. The Top End RHD control program can be used as a model for other RHD programs. Other states and territories in Australia with high rates of ARF and RHD should implement RHD control programs.

Introduction

Rheumatic heart disease (RHD) continues to be a significant health problem for many Aboriginal people living in the Northern Territory (NT).[1,2] Research by the Menzies School of Health Research (MSHR) has demonstrated that the NT has amongst the highest published rates of acute rheumatic fever (ARF) and RHD in the world.[1-4] In November 1997 the Centre for Disease Control (CDC), Territory Health Services (THS) and MSHR established a RHD control program in the Top End of the NT. Funding for the program was obtained from the Australian Institute of Health and Welfare and the Commonwealth Department of Health and Family Services.

The objectives of this program are:

1. to create and maintain a computerised register of people in the Top End with known or suspected ARF and RHD,
2. to develop methods of improving adherence to ongoing secondary prophylaxis,
3. to provide appropriate health education for people with ARF and RHD and their families and carers,
4. to provide communication with and support to health staff working in remote areas,
5. to standardise the clinical care of people in the Top End with ARF and RHD according to best practice,
6. to provide prospective epidemiological data to monitor the incidence and prevalence of ARF and RHD in the Top End.

In this paper we describe progress on the first five objectives. Disease rates are described in the following paper.[5]

Objective 1 - To create and maintain a computerised register of people in the Top End with known or suspected ARF and RHD

An existing rheumatic heart disease research database established by Jonathan Carapetis at the MSHR was converted from Oracle to Microsoft Access. Fields pertaining to research were omitted. Additional fields useful for service delivery, including a recall system, were added. Data collected and stored for individuals on the register now include: demographic information, diagnostic history, secondary prophylaxis use and adherence, heart valve surgery profile, clinical progress in-

cluding cardiac status, and the need for ongoing care. Additional information such as surgery waiting lists, and the date and cause of death of people with RHD is also recorded.

There continues to be a number of people who are unknown to the register but have established RHD. These people are identified in a number of ways. Copies of specialist review letters and echocardiograms of people with established heart disease are automatically forwarded to the register. Unregistered people are identified through the THS hospital information system, through liaison with the community health centres, and by direct discussions with treating health staff, and then added to the register. Updated demographic and medical information is recorded on the register, and computerised recall function is activated. Recall either defaults to best practice treatment guidelines or specific requests by the medical specialist, depending on current cardiac status and previous follow-up.

The register is currently operated manually and the fields have changed many times during the establishment of the program. An automated system is now planned with the use of forms within the database. This will provide regular reports automatically.

The RHD control program computerised register is strictly confidential and has password-only access. It is accessed only by the RHD program coordinator. Individual patients are identified only where it is necessary for their ongoing medical care. Information is passed only to health staff on a need to know basis directly related to individual patient care. The data fields on the register relate only to the identification and residence of patients and to their ARF and RHD. The register is subject to the THS information privacy code of conduct. A steering committee has been formed involving members of the Aboriginal community, rural and remote health staff, public health specialists, specialist physicians, the National Heart Foundation and members of the Commonwealth funding body. This committee meets every 2 months.

Objective 2 - To develop methods of improving adherence to ongoing secondary prophylaxis

To assist in the identification and evaluation of adherence at community level, 4 target communities were identified and provided with active assistance for secondary prophylaxis adherence. Intervention, including community-based education for both patients and staff, and discussions to identify strategies to improve adherence, was conducted in these four communities. The main aim was to increase the number of benzathine penicillin injections received, thus reducing ARF recurrences, which leads to cumulative valve damage.

Specialists have been asked to comment on adherence to secondary prophylaxis medication during routine patient review, and this information is updated on the register against the patient records. Patients seen in hospital during acute episodes of ARF are encouraged to adhere to secondary prophylaxis. A needle chart to record monthly injections is offered to the patient/family.

We planned to measure community and individual adherence to long acting benzathine penicillin in 1997 and 1998 (by calculating the number of injections received, out of a possible 12 per calendar year). However, this data is not reported here because information on many patients was missing or incomplete.

A number of communities have preferred not to provide copies of 'Bicillin lists', and not all visiting specialists comment on recent adherence to medication during routine reviews. The program is working on strategies to improve this situation with respect to data.

Adherence to secondary prophylaxis is an issue that requires more involvement with remote Aboriginal health workers, treating medical officers, nursing staff and clients with rheumatic heart disease. High staff turnover, community activities and priorities, the movement of people between communities and regional centres and the demanding workloads at community clinics all contribute to often inadequate levels of secondary prophylaxis adherence. High staff turnover in the communities and empowerment of Aboriginal health workers to take initiatives for promoting care are currently being addressed. The rheumatic heart disease control program will continue to work to promote sustainable practices.

Objective 3 - To provide appropriate health education for people with ARF and RHD and their families and carers

RHD information packages were forwarded to all

community health centres and Aboriginal Medical Services in the NT. This information package contained a copy of the RHD booklets, needle chart and video (produced by the RHD team at MSHR), and a copy of the NT standard treatment guidelines for adults with ARF and RHD. Additional copies of the booklets and video are available free for patients through MSHR.

Aboriginal health workers based at Royal Darwin Hospital (RDH) provide patient education during acute episodes of ARF. In addition, an Aboriginal health worker from the MSHR has concentrated on the education of children suffering from ARF during acute admission, and has produced a number of interactive education tools.

Disease education for new and existing health staff is provided through the: THS Staff Development Branch, the education coordinators within RDH, and Aboriginal Health Worker training facilities. Education is conducted at RDH, and opportunistically at Gove, Katherine and Alice Springs hospitals.

Objective 4 - To provide communication and support to health staff working in remote areas

During the program establishment phase, a letter of introduction was sent to Aboriginal medical services staff, THS staff and all remote communities in the Top End. This generated interest and feedback about the program, and prompted staff to raise issues about RHD and ongoing treatment. The program has increased awareness of the disease, and promoted the importance of efficient diagnosis and notification of episodes of ARF and established RHD. This has led to the identification and addition to the register of a large number of previously unknown people.

Travel to communities by the program coordinator has remained a priority of the program. Issues are discussed with health staff at community level, and strategies for improving secondary prophylaxis adherence are tailored to each community setting. Community profile reports are regularly generated from the register and provided for district and local medical officers, as well as health centre nursing and Aboriginal health staff.

Information on patient status and outstanding treatment is fed back to the communities at various levels. Lists of specialist reviews that are due/ overdue are sent to the specialist responsible for care, as well as the community (medical officer or nurse/health worker). 'RHD reports' are also generated for the communities. These reports provide information on total numbers of people on the register from the community and their diagnosis, a current benzathine penicillin list, relevant diagnostic and surgical history, a general recall list (projecting 2-3 months to assist with the planning of care), and any other relevant information. A number of remote district medical officers have commented favourably on the ability of the reports to provide a cross-check for both the register and the primary health care staff at community level. These reports also provide up-to-date information for new health staff (as there are problems with high staff turnover in remote areas).

Objective 5 - To standardise the clinical care of people in the Top End with ARF and RHD according to best practice

People on the register become lost to follow-up for a number of reasons. The register generates a regular list of people with established heart disease who have not been medically reviewed, or presented to a NT hospital within a certain time. Communities where the person is known to live or visit are contacted in an attempt to locate the person and inform them of the need for ongoing management.

The register is able, via the various feeder and information systems, to identify new and existing cases of ARF and RHD in the Top End. Currently, an average of 2-3 people are added to the register each week. As of 1 June 2000 there are 580 people with established RHD on the register.

The register is able to help identify people who require an echocardiogram. When faced with limited time or resources, the register, in liaison with the community medical officer, has been able to assist with the prioritisation of people according to their cardiac status, and possible need for surgery. It has been noted that patients have occasionally been identified for heart valve surgery, and for a number of reasons surgery has not been booked. The register has thus established a surgery recall list. Dates for travel and procedures are updated as they are identified. This information is fed back to physicians and remote clinics regularly. Surgery waiting lists are discussed regularly with primary health care staff, treating physicians and NT Cardiac Services.

Conclusions

The RHD control program has achieved many of its original objectives. The Top End program has also been able to share its experience and knowledge with Central Australia, the Kimberley region of Western Australia and North Queensland in their efforts to develop plans for their own RHD control programs. We plan to integrate the program more fully into routine services in the NT in the coming years.

Acknowledgements

We would like to thank the Australian Institute of Health and Welfare and the Commonwealth Department of Health and Family Services for start-up funding of the Top End Rheumatic Heart Disease control program. We would also like to thank Geoff Angeles and Loyla Lesley from MSHR for developing the RHD education materials and the National Heart Foundation and our clinical colleagues in the NT for their ongoing support of the program.

References

1. National Centre for Monitoring Cardiovascular Disease. Heart Stroke and Vascular Diseases. Australian Institute of Health and Welfare and Heart Foundation of Australia May 1999. AIHW Cat. No. CVD 7.
2. Carapetis, JA, Wolff DR, Currie BJ. Acute rheumatic fever and rheumatic heart disease in the Top End of Australia's Northern Territory. Med J Aust 1996;164:146-149.
3. Carapetis, JA, Currie BJ. Preventing Heart Disease in Australia. Med J Aust 1998; 168: 428-429.
4. World Health Organisation. Rheumatic fever and rheumatic heart disease: report of a WHO study group. World Health Organisation Tech Rep Ser 1988:764.
5. Edmond KM, Noonan S, Krause VL, Currie BJ. The Top End rheumatic heart disease control program II. Rates of rheumatic heart disease and acute rheumatic fever. NT Disease Control Bulletin June 2001; 8(2).

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The Top End rheumatic heart disease control program
II. Rates of rheumatic heart disease and acute rheumatic fever

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Abstract

Objective: To describe the incidence and prevalence of acute rheumatic fever (ARF) and rheumatic heart disease (RHD) in the Top End of the Northern Territory (NT) from January 1997 to December 1999 and to compare with previously published data.

Methods: Notifications of RHD and ARF were collated prospectively from 1 November 1997 to 31 December 1999. Data was retrospectively collected from 1 January 1997 to 31 October 1997. Population denominators were obtained from 1996 Australian Bureau of Statistics census data. Annual ARF incidence, proportion of recurrent ARF and point prevalence of RHD were calculated.

Results: Thirty-eight non-Aboriginal people had RHD in 1999 (point prevalence 0.34/1,000) but none had ARF. RHD in Aboriginal persons of all ages has increased from a point prevalence of 8.5/1,000 in 1988 to 13.3/1,000 in 1999. Total rates of ARF are unchanged (84.3/100,000 in 1994, 89.5/100,000 in 1999) but recurrent ARF (as a proportion of total ARF notifications) decreased to 16% in 1999.

Conclusions and implications: The increasing rates of RHD and ARF in the Top End are most likely due to increased ascertainment rather than an increase in actual disease. If the RHD control program is to have a sustainable impact, further reduction in the proportion of recurrent ARF should occur, followed by decreasing annual rates of ARF and new diagnoses of RHD. We will then begin to see a decrease in the total number of RHD cases in the Top End of the NT.

Introduction

Rheumatic heart disease (RHD) continues to be a significant health problem for many Aboriginal people living in the Northern Territory (NT).[1,2] High recurrence rates of acute rheumatic fever (ARF) and difficulties with adherence to benzathine penicillin prophylaxis are daily management problems. Research by Carapetis and the Menzies School of Health Research (MSHR) has demonstrated that the NT has amongst the highest published rates of ARF and RHD in the world.[1-4] The World Health Organisation recommends that all areas with high rates of ARF and RHD institute a coordinated, register-based control program.[4] In November 1997 the Centre for Disease Control (CDC), Territory Health Services (THS) and MSHR established a RHD control program in the Top End of the NT. Start-up funding for the program was obtained from the Australian Institute of Health and Welfare (AIHW) and the Commonwealth Department of Health and Family Services (CDHFS).

The objectives of this program are described in the accompanying article.[5] In this paper we report on incidence and prevalence data collected by the control program from January 1997 to December 1999.

Methods

Design

This was a prospective population based study of all notifications of RHD and ARF from 1 November 1997 to 31 December 1999. Data was retrospectively collected from 1 January 1997 to 31 October 1997.

Setting

The Top End of the NT consists of 3 administrative regions: Darwin, Katherine and East Arnhem. Each region is served by 1 hospital with the RDH acting as the tertiary referral centre. The total population of the Top End according to 1996 census data is 147,931 of whom 47% are female and 25% Aboriginal.[6]

Case definitions

RHD was defined as the presence of a heart valve lesion thought to be due to previous ARF based on clinical or echocardiographic evidence. An episode of *initial ARF* was defined according to the 1992 revised Jones criteria (see table 1).[7] A *recurrence of ARF* was defined as symptoms or signs consistent with Jones' criteria occurring greater than 3 months after an acute episode for which there was documented evidence of recovery.[7] Incident cases were defined according to date of onset of symptoms or signs. *Total ARF* was defined as initial plus recurrent episodes of ARF in 1 year. *Possible ARF* was defined as fever (> 37.5°C) and monoarthritis/polyarthritis or a new murmur consistent with ARF plus evidence of recent group A streptococcal infection. Cases of possible ARF were not used to calculate incidence rates but they were monitored for development of ARF or RHD.

Table 1 Criteria needed for the diagnosis of acute rheumatic fever[7]

Two major revised Jones' criteria or 1 major & 2 minor	
Major Jones' criteria	Minor Jones' criteria
carditis	arthralgia
polyarthritis	fever (> 37.5° C)
chorea	Raised ESR or CRP
Erythema marginatum	Prolonged PR interval
Subcutaneous nodules	
and	
Evidence of recent group A streptococcal infection	
(positive throat culture, anti-streptolysin O antibody titre > 256IU or anti-DNAase B > 300IU)	

Population denominators

Denominators were calculated from 1996 census data, which includes experimental population estimates for the Aboriginal population.[6] The mid year estimated populations for 1997 to 1999 were calculated from this census data.

Data collection

ARF became a notifiable disease in the NT in 1994. Data on ARF and RHD has been recorded in the RHD control program computerised register since November 1997. All health care providers in the NT are asked to notify their local CDC about all cases of ARF and RHD. In addition, all physicians and paediatricians in the Top End of the NT provide the RHD control program with correspondence and echocardiogram reports that document ARF or RHD. New information about existing cases is also obtained from hospital records and health staff.

Data was obtained for 1 January 1997 to 31 October 1997 by reviewing the CDC notifiable disease database and by contacting all physicians, paediatricians and remote area medical officers in the Top End of the NT. In some cases hospital files were consulted.

The following data was collected: name, date of birth, community of residence, hospital registration number, date of notification, date of diagnosis, preceding sore throat, type of ARF (carditis, chorea, arthritis, erythema marginatum, subcutaneous nodules), recurrent ARF, possible ARF, type of RHD, echocardiogram result, hospitalisation, number of benzathine penicillin injections per year, medical review and surgical review.

Data analysis

Annual incidence of ARF was calculated for all incident cases of ARF (initial and recurrent) using the mid year estimated population obtained from the 1996 census.[6] Average annual incidence was calculated for years 1994-1999. Rates were also calculated for Aboriginal and non-Aboriginal subgroups and for age groups 0-4, 5-14 and $\geq$ 15 years. Proportion of recurrent ARF was calculated using the number of cases of recurrent ARF in one year as the numerator and total incident ARF (initial + recurrent) as the denominator. Proportions were also calculated for Aboriginal and non-Aboriginal subgroups and for age groups 0-4, 5-14 and $\geq$ 15 years. Point prevalence of RHD was calculated for October of each year (to allow comparability with previous data[2]) using the number of existing cases of RHD as the numerator and the annual mid year estimated population as the denominator. Rates were also calculated for Aboriginal and non-Aboriginal subgroups and for age groups 0-4, 5-14 and $\geq$ 15 years.

Results

Incidence of ARF and point prevalence of RHD for Aboriginal people of all ages and Aboriginal school children are shown in tables 2 and 3 and in figure 1. Thirty-eight non-Aboriginal people had RHD in 1999 (point prevalence 0.34 / 1,000) but none had ARF. From 1994 - 1999 Aboriginal school children accounted for almost two thirds of all cases of ARF among Indigenous people in the Top End (107 cases). The average annual incidence of ARF in children aged 5-14 years appears to have decreased from 1988 to 1999. For all age groups recurrent ARF has decreased from 10 cases (28% of total ARF) in 1998 to 6 cases (16% of total ARF) in 1999, with only 1 child being notified with recurrent ARF in 1999. We also report 23 cases of possible ARF (8 cases in the 5-14 year age group) from 1998 and 1999. In 1999, the prevalence of RHD was 13 times higher in Indigenous people (n=490) compared with non-Indigenous people (37 cases) and there were 49 school-aged children with RHD.

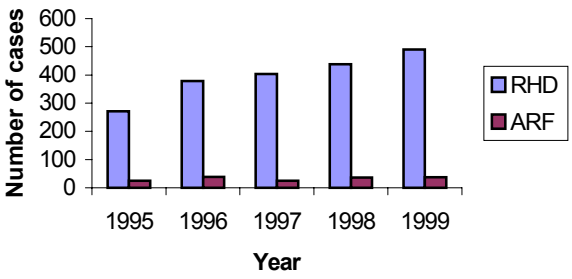
Table 2 Acute rheumatic fever among Aboriginal people in the Top End of the NT 1988-99

	5-14 years		All ages	
Years	Rate per 100,000 population	No.	Rate per 100,000 population	No.
1988-93[2]	254	91	-	-
1994[2]	204	18	84.3	27
1995[2]	148	13	78.0	25
1996[2]	238	21	105.0	38
1997	159	14	69.0	25
1998	270	24	101.0	36
1999	191	17	100.0	37
1994-1999	202	107	89.5	188

Table 3 Rheumatic heart disease amongst Aboriginal people in the Top End of the NT 1995-99

	5-14 years		All ages	
Years	Point prevalence per 1,000 population	No.	Point prevalence per 1,000 population	No.
1995[2]	3.5	31	8.5	271
1996[2]	4.3	38	10.4	378
1997	4.0	35	11.1	403
1998	4.6	41	12.2	438
1999	5.5	49	13.3	490

Figure 1 Total number of Aboriginal people from 1995 to 1999 in the Top End of the NT with acute rheumatic fever or rheumatic heart disease



Discussion

The increasing rates of RHD and ARF in the Top End are most likely due to increased ascertainment rather than an increase in actual disease. The incidence rates published by Carapetis et al in 1996 underestimate the true burden of RHD and ARF.[2] At least 200 new cases of ARF or RHD have been notified to the RHD control program since that study was published. Approximately 2 cases of previously undiagnosed RHD are notified each week to the RHD control program coordinator and an average of 3 cases of ARF are notified each month (personal communication, Sara Noonan).

We report very high rates of ARF in children aged 5-14 years. The rates appear to be decreasing but still far exceed rates of RHD or ARF reported in poor areas of urban Australia 50 years ago.[1,2,8] The problem of ARF in school-aged children is well known.[8-10] Close physical contact, high group A streptococcal colonisation and high transmission rates are contributing factors.[4,8-10] Recurrent ARF appears to be decreasing. This may indicate that benzathine penicillin prophylaxis use is improving. Information on secondary prophylaxis and health education is described in the accompanying article.[5]

New data from the AIHW indicate that hospitalisation rates are very high for Aboriginal Australians with ARF (Aboriginal people account for 14% of the hospitalisations for ARF and RHD).[1] Of the hospitalisations for ARF, 54% occur amongst those aged 5-19 years. Other data from the AIHW demonstrates that deaths attributable to RHD or ARF are declining at a rate of 3.9% per year for males and 4.1% per year for females.[1] However, one third of deaths still occur in persons under 70 years of age. The AIHW data also indicates that Indigenous Australians are 16 times more likely to die from ARF or RHD than other Australians and the death rate attributable to ARF or RHD is higher in remote areas than urban centres.[1]

Rates of ARF and RHD are high in other parts of Northern Australia and Central Australia.[11-13] A RHD control program will be commencing in Central Australia and there are initiatives underway in North Queensland (Dallas Young and Jeffrey Hanna, personal communication) and the Kimberley region of Western Australia (Donna Mak, personal communication).

There is still much work to be done in improving the rates of ARF and RHD in the Top End of the NT. We will continue to produce yearly epidemiological reports. This information will be used to lobby for more resources for primary prevention of RHD and ARF. We will also continue to: maintain the computerised RHD register, develop and trial methods of improving adherence to secondary prophylaxis, assist in coordinating the clinical care of persons with RHD or ARF, assist in improving the clinical care of people in the Top End with ARF/RHD according to best practice and will continue to provide appropriate health education for people with ARF/RHD and their families and carers. We will also continue to advocate for improved primary health care services and for initiatives to improve the socio-economic and educational disadvantage in remote Aboriginal communities. These factors are the major determinants of the high rates of ARF and RHD.[14] Sustainable impact will be reflected in a decrease in the proportion of recurrent ARF (rather than first episodes), followed by decreasing annual rates of ARF and new diagnoses of RHD. We will then begin to see a decrease in the total number of RHD cases in the Top End of the NT.

Acknowledgements

We would like to thank the Australian Institute of Health and Welfare and the Commonwealth Department of Health and Family Services for start-up funding for the Top End Rheumatic Heart Disease control program. We would also like to thank all of our clinical colleagues for supporting the program.

References

1. National Centre for Monitoring Cardiovascular Disease. Heart Stroke and Vascular Diseases. Australian Institute of Health and Welfare and Heart Foundation of Australia May 1999. AIHW Cat. No. CVD 7.
2. Carapetis, JA, Wolff DR, Currie BJ. Acute rheumatic fever and rheumatic heart disease in the Top End of Australia's Northern Territory. Med J Aust 1996;164:146-149.
3. Taranta A, Markowitz M. Rheumatic fever. Boston: Kluwer Academic publishers, 1989:1-29.
4. World Health Organisation. Rheumatic fever and rheumatic heart disease: report of a WHO study group. World Health Organisation Tech Rep Ser 1988:764.
5. Noonan S, Edmond KM, Currie BJ, Krause VL. The Top End rheumatic heart disease control program I. Report on program objectives. NT Disease

Control Bulletin June 2001; 8(2).
6. Australian Bureau of Statistics. Population by age and sex, Australian states and territories. Canberra 1996, catalogue #3201.0.
7. Special writing group of the committee on Rheumatic fever, Endocarditis and Kawasaki Disease of the Council on Cardiovascular Disease in the Young of the American Heart Association. Guidelines for the diagnosis of rheumatic fever. JAMA 1992;268:2069-2073.
8. Holmes MC, Rubbo SD, A study of rheumatic fever and streptococcal infection in different social groups in Melbourne. J Hyg 1953;51:450-7.
9. Chun LT, Venu Reddy D, Yamamoto LG, Rheumatic fever in children and adolescents in Hawaii. Pediatrics 1987;79:549-52.
10. Jose DG, Welch JS, Studies of Australian Aboriginal children; streptococcal infection, heart reactive antibody and subclinical rheumatic carditis. Aust Paediatr J 1969 5:209-18.
11. Brennan RE Patel MS Acute rheumatic fever and rheumatic heart disease in a rural central Australian community. MJA 1990: 153: 335-9.
12. Patten BR. Rheumatic fever in the West Kimberley MJA Spec Suppl 1981;1:11-15.
13. Richmond P, Harris L. Rheumatic fever in the Kimberley region of Western Australia. J Trop Pediatr 1998;44:148-52.
14. Carapetis J, Currie BJ. Preventing rheumatic heart disease in Australia [editorial]. Med J Aust 1998;168(9):428-9.

Editorial comment

Tarun Weeramanthri, CDC Darwin

The above articles report on the Top End Rheumatic Heart Disease Control Program, which was established in late 1997. Progress has been impressive in collecting clinically relevant information in a data system that is then used to coordinate follow up (especially with respect to the timing of surgery if needed), but more resources are needed to make a sustained impact on adherence to penicillin prophylaxis (the most crucial intervention in preventing progression of disease). In the last year, Central Australia has also established a similar program. Meanwhile, management of acute rheumatic fever and rheumatic heart disease are highlighted in the CARPA and GSAT Guidelines, and health professional education and auditing will be carried out by the Total Recall nurses in the Top End. However, due to resource constraints, the Program Coordinator in the Top End is now employed for only 1 day a week. We are currently negotiating with other stakeholders for funds to sustain the program in the Top End and Centre and expand its community education component.

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NT Disease Control Workshop

18 - 20 September 2001

Mirambeena Resort, Darwin

The annual Disease Control Workshop is attended by CDC representatives from all NT districts and a variety of health professionals and interested individuals. A range of topics are presented covering both local and broader issues.

This year Dr Hume Field, sponsored by CSL, will present his work on bats and emerging diseases. Dr Field is the Principal Veterinary Epidemiologist (Emerging Diseases), Animal & Plant Health Service, Department of Primary Industries, Qld.

All interested parties are invited to attend.

For further details call CDC Darwin on 08 892 28044

The Population Health Education for Clinicians Project – are you interested in participating in a trial run?

Paul Kelly[1,2], Simon Morgan[3], Fay Johnston[4], David Peacock[2], Chris Harrison[4] and Ross Bailie[1,5].
Menzies School of Health Research, Darwin[1], Centre for Disease Control, Darwin[2], General Practice Education and Research Unit, Darwin[3], Top End Division of General Practice[4], Flinders University NT Clinical School[5]

Background

In 1998, the Commonwealth Department of Health and Aged Care (DHAC) funded a series of initiatives aimed to increase the capacity and willingness of General Practitioners (GPs) to participate in Population Health initiatives. These have included the formation of a Joint Advisory Group (with the National Public Health Partnership and the General Practice Partnership Advisory Council), practice incentive payments for population health activities in General Practice and the Population Health Education for Clinicians (PHEC) project. The PHEC was conceived after extensive nationwide consultation revealed that there was a perceived need by practicing GPs for a post graduate qualification in population health. The consultation group concluded that this need was not fulfilled by currently available Masters of Public Health programs offered through universities.

The PHEC represents a collaboration between the Royal Australian College of General Practice (RACGP), the Australian College of Rural and Remote Medicine (ACRRM), Divisions of General Practice and University Departments of Rural Medicine, General Practice and Public Health. The coordinating group set out a wide range of competencies and arranged them into three clusters of four modules. The modules are based on clinical groupings that, for the most part, reflect national priority areas in population health and general practice. Eight universities accepted the invitation to write the 12 modules (see table 1).

Target groups

The PHEC modules are primarily designed for practicing GPs. However, other groups may be interested in the modules, including GP Registrars, other clinicians, AFPHM fellows and trainees and other health professionals (particularly remote nurse practitioners). Individual modules will contribute to CME points for qualified GPs. Completion of a cluster fulfils a set of selected competencies in population health. Integration of modules into existing awards will be possible at participating universities. Ideally, clinicians would complete all 12 modules and gain a formal postgraduate qualification in population health.

Table 1 The clusters and modules of the PHEC

Cluster No.	Cluster name	Module name
1	Practice evaluation and research	Cardiovascular disease
		Cancer
		Occupational health & safety
		Obesity
2	Population health strategies	Mental illness
		Chronic & complex diseases
		Diabetes
		Infectious diseases
3	Health promotion & policy	Aboriginal health
		Drugs & alcohol
		Nutrition
		Health Promoting Medical Practice

The modules

Each module is designed to be completed in approximately 40 hours, including readings, learning activities and assignments. The modules follow a similar template starting with a clinical "case scenario" (or series of scenarios) to place population health activities within settings familiar to practicing clinicians.

The Infectious Diseases module

In September 2000, in recognition of their interest and expertise, Menzies School of Health Research was approached to write the infectious disease module. In November, a committee was formed consisting of population health and general practice academics, practicing GPs and public health physicians.

The committee had 4 guiding principles:

1. to fulfil the competencies as outlined in the module brief;
2. to concentrate on clinical scenarios which were common and/or reflected national priority diseases;
3. to keep the content relevant for GPs;
4. to use entirely web-based resources and references.

Whilst the content had a distinct flavour of rural/ remote and Aboriginal health issues, reflecting the context in which the authors work, the module was written with a view to appealing to a wider audience.

The phases of development of the ID module included:

1. formulation of case scenarios based on the clinical experience of the group;
2. development of learning objectives based on the module competencies;
3. matching of the learning objectives to the case scenarios;
4. creating assessments which related to the learning objectives;
5. independent review of the module;
6. refinement of the content and presentation of the module.

The paper based product is now complete and web-based and CD ROM versions are under development. The final product will be available for trialing by the end of July, 2001.

Implementation

A series of nationwide trials of the modules are planned to coincide with the second University semester of 2001. Four geographically based consortia have been formed (Queensland, New South Wales, Victoria and South Australia/Northern Territory/Western Australia) with Universities collaborating to deliver two clusters in each region. A variety of modes of delivery will be used in the trial including web-based, CD-ROM, workshops and paper based distance education. Incentives for participants in the trial include credit for University postgraduate qualifications, Continuing Medical Education points, scholarships (fee or HECS waived) and remuneration. For the trials, the consortia are interested in hearing from people who are prepared to complete a cluster (that is four modules). Participants who enrol in a cluster will be required to complete approximately 160 hours of coursework, submit assignments and participate in the formal evaluation process for the trial. It may also be possible to enrol in individual modules. Potential trials in Darwin include workshops for qualified GPs (leading to CME points) and for GP registrars.

The aim is for full implementation of the PHEC modules in 2002, with several Universities offering the modules as Graduate Certificates in Population Health.

For more background on PHEC, see www.cme.net.au/phec/index.htm

If you are interested in participating in a trial, contact Paul Kelly (paulk@menzies.edu.au).

Acknowledgements

Department of Health and Aged Care for funding the project, and the many collaborators working on the PHEC writers' forum who have offered valuable feedback about the Infectious Disease module. Justine Mayer of TEDGP also gave a considered appraisal of an early draft of the module.

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The risk of relapse of multibacillary leprosy after multi-drug therapy

Nathan Zweck, CDC, Darwin

Introduction

Although the incidence of leprosy in the NT is now very low, the disease contributes importantly to the Centre for Disease Control's (CDC) scheduled workload through the annual follow up of previously treated cases *for life*, and their contacts for *20 years*. These are the recommended durations in the 'Guidelines for Leprosy Control in the Northern Territory', last revised in 1996. For example, in the East Arnhem district alone, 317 such individuals are on annual follow up lists, comprising 98 indexes, 119 household contacts, and 100 others under surveillance with possible signs of leprosy. Relapses and late reactions can occur many years after treatment is completed, with the possible consequences of further disability for individuals, and fresh transmission of leprosy in communities.

The reality however is that CDC staff find it difficult to find a place consistently for this follow up amongst many other disease control priorities. There is a need to target for annual follow up the fraction of these individuals who are at *highest* risk of relapse because of disease type, or at uncertain risk after treatment with non-multi-drug therapy (MDT) regimens, and who therefore warrant serial skilled examinations and smears. Others at lower risk may then be discharged to the care of local health staff, with education about the signs of relapse and reaction, and the need to represent if they occur.

A recent editorial in the *Leprosy Review*[1] has prompted a review of this subject.

Relapse rates after MDT

In the early 1990s, confidence in the efficacy of WHO's MDT regimens for Pauci- (PB) and Multi-bacillary (MB) forms of leprosy was based on low levels of reported relapse after treatment in a large multi-centre survey. Following treatment of 20,141 MB patients treated and followed from 1984 to 1992 there were only 67 cases of relapse (cumulative risk 0.77%), with proportions of 0.25% in the first 3 years of follow up, 0.29% during 4-6 years, and 0.22% during 7-9 years[2]. So low were these figures, that earlier WHO recommendations for control programs to annually review all leprosy patients after release from treatment (PB for 2 years and MB for 5 years)[3] were withdrawn in 1994[4]. One conclusion was uneqivocal:

"With such a low risk for relapse and since the majority occur within a few years after stopping MDT, there is definitely no need to have long-term active post-MDT surveillance of patients for the purpose of detecting relapse".[2]

The first real qualms arose in 1995 with the report of Jamel and Ji and the Marchoux Chemotherapy Study Group who showed a 20% relapse rate in a small sample of MB patients followed for 7 years post-treatment[5]. All relapses occurred in the subset with a high Bacillary Index (BI) of >= 4. The BI is a logarithmic scale from 0 to 6+ which represents the numbers of *M.leprae* seen on microscopy of skin smear specimens. A number of subsequent small-scale studies of patients treated with MB-MDT (where relapse rates were calculated for high- and low-bacillated groups) have produced conflicting conclusions [5-11]. (Tables 1 & 2).

Table 1 Studies suggesting high rates of relapse associated with high BI after MB-MDT

Authors	Sample	Duration of MDT	Overall MB relapses	Relapses B I >=4	Relapses BI <4	Follow up period (years)	Definition of relapse
Jamet et al[5] (Mali) 1995	35	24 months	7/35 (20%) **3.3 /100py**	7/18 (39%) **7.0 /100py**	0/17 (0%) **0 /100py**	mean 6 +/- 1.4	BI increase by 2+ over previous value at any single site of old lesions & New lesions with higher BI than any pre-existing lesion
Girdhar et al[6] (India) 2000	260	24 months	20/260 (8%) **2.0 /100py**	18/107 (17%) **4.3/100py**	2/153 (1%) **0.4/100py**	3.7	BI increase by >=2 at any site
Girdhar et al[6] (India) 2000	301	Until smear negative	12/301 (4%) **1.1 /100py**	11/258 (4%) **1.3 /100py**	1/43 (2%) **0.5/100py**	mean 4.9 +/- 2.3	BI increase by >=2 at any site

Table 2 Studies suggesting low rates of relapse associated with high BI after MB-MDT

Authors	Sample	Duration of MDT	Overall MB relapses	Relapses BI >=3	Relapses BI <3	Follow up period (yrs)	Definition of relapse
Jesudasan et al[7] (India) 1996	261	24 months	0/261 (0%) **0/ 100py**	0/34 (0%) **0 /100py**	0/227[8] (0%) **0/ 100py**	mean 6.1 +/- 2.1	not stated
Jesudasan et al[9] (India) 1996	505*	Until smear negative	0/505 (0%) **0 /100py**	0/35 (0%) **0 /100py**	0/470 (0%) **0 /100py**	mean 10	Increase in the BI of 1+ or more at any site on 2 consecutive smears at a 6-month interval
Shaw et al[10] (India) 2000	46**	24 months		1/46 (2%) **0.23/100py**		minimum 5 mean 9.2 +/- 3	Increase in the mean BI of 1+ or more with or without clinical signs of activity
Gebre et al[11] (Ethiopia) 2000	256	24 months	0/256 (0%) **0 /100py**	0/57 (0%) [BI >=4] **0 /100py**	0/199 (0%) [BI <4] **0 /100py**	mean 4.3	Smear with Morphological Index*** of >=2% or BI increase by >=2 in 2 sites

* 96% had been treated previously with Dapsone monotherapy
** includes the 34 with BI >=3 in Jesudasan et al[7] above
*** percentage of solid staining bacilli (indicating live bacilli)
Note: py = person years

Discussion

These results have limited application to the situation in the NT since the treatments that have been applied in the NT have not been classical WHO MB-MDT. The current NT regimen for MB leprosy differs from the WHO treatments studied above in it's prescription of rifampicin daily rather than monthly. Thus, the relapse rates in the above studies cannot necessarily be extrapolated to the NT regimen. Intuitively, one would expect the NT regimen with 30 times the total dosage of rifampicin to be more efficacious and associated with fewer relapses, but this is not known. Older regimens in the NT were various combinations of one to three anti-leprosy drugs for varying durations – most lepromatous patients who had received monotherapy in the NT were re-treated with 3 months of daily Rifampicin, or multi-drug regimens. There are insufficient studies of long-term relapse rates after these combinations. It is known however that dapsone monotherapy, employed world-wide until the 1960s, was associated with around a 10% relapse rate[12].

Some points however can inform our guidelines. After fixed duration therapy, highly bacillated MB patients are a high risk group for relapse in settings *where relapses have occurred*[5,6]. The relapses may not occur until 5-7 years after treatment completion[5], although Girdhar's relapses were detected within 3 years of follow up[6]. Taking a conservative line until better evidence is available would mean *continuing* to identify this highly bacillated group by the use of skin smears at diagnosis. (There has been a tendency for large control programs to abandon the skin smear which is painful for patients, and time-consuming to perform and for the laboratory to read, in favour of a simple clinical classification of patients as PB or MB.) In the NT where the new patient load has not been high in recent years, we should continue to smear all new patients, and annually follow post-treatment those with a pre-treatment BI of >=4 for 10 years with a clinical examination for new lesions and skin smears. Annual review by an ophthalmologist should also be organised for this group to prevent blindness caused by relapse or reaction in the anterior segment[13].

In our setting, ways could be explored to reduce the pain for patients who undergo a smear. Recently, pre-treatment of the skin with EMLA topical anaesthetic cream provided effective analgesia to deep pin-prick 1 hour later on my ear lobe. It needs to be determined whether smear quality is compromised by the cream or some other factor. Infiltrating with subcutaneous local anaesthetic is not appropriate due to it's dilutional effect on the smear with false negative results a possibility.

Even in the studies in Table 1, it appears that relapse rates are low enough in those with BI <4 to allow these patients to be discharged after MDT to the care of their local medical service without annual smears and examination by CDC being required. The larger concern in this group is acute nerve damage due to Type 1 reactions in the first 3 years after treatment completion. Croft points out that routine annual surveillance has only a 50% chance of detecting such damage that is less than 6 months old and thus reversible with corticosteroid treatment[14]. He found that health education about the signs of relapse and reactions given at release from treatment was effective in motivating patients to *self-report*, and that many more reactions were detected by self-reporting than by even 3-monthly routine surveillance[14].

For those in the NT who were treated more than 10 years ago with regimens of different drug combination, dosage, or duration than the current ones, it seems reasonable to follow all the MB patients for life, since the long-term relapse rates of these regimens is not known and may be higher than those quoted for modern MDT. The PB patients could now be discharged to the care of the general health services.

Contacts of highly bacillated (BI >=4) MB patients could be followed annually with a clinical examination for 10 years. There is some evidence that 95% of secondary leprosy cases will be diagnosed within 6 years of the diagnosis of the index[15]. Contacts of other MB patients and PB patients could be assumed to be at lower risk and discharged after an initial examination looking for a source index, and training which will lead to self-reporting of signs.

Conclusions

The implication of the discussion above is a large reduction in workload for CDC staff, and an increased probability of annual review for those who really require it. In East Arnhem the follow up list would reduce from 317 to 58 (24 indexes and 34 contacts). Even if all 58 could not be reached annually, staff could prioritise them in descending order of their initial BIs (ie review 6+ first, then 5+), or clinical classification (ie LL first, then BL), and so on. Priorities could be further stratified within these BI groups according to whether or not rifampicin had been received over 24 months.

The studies above look at several hundred patients only, when millions more have been treated with MDT globally. Evidence based on larger sample sizes with follow up periods beyond 10 years can be expected in the next decade and will further enlighten our practice in the NT. While any doubt remains about the long term efficacy of fixed-duration MDT for 24 months for highly-bacillated MB leprosy, it would be unwise to move to 12 month durations being implemented elsewhere. Finally, if we wish to apply global research about MB-MDT treatment to our program, consideration should be given to adopting the WHO regimen (monthly Rifampicin).

The 'Guidelines for Leprosy Control in the Northern Territory' are presently under review for updating by the end of 2001. Please contact Nathan Zweck (89228898 or nathan.zweck@nt.gov.au) if you are interested in contributing to this process.

References

1. Ji B. Does there exist a subgroup of MB patients at greater risk of relapse after MDT? *Lepr Rev*, 2001; **72**, 3-7
2. WHO Leprosy Unit. Risk of relapse in Leprosy. *WHO document WHO/CTD/LEP 94.1*
3. WHO. *A Guide to Leprosy Control* 2nd ed., Geneva 1988, p.88
4. WHO. *Chemotherapy of Leprosy*, WHO Technical report series 847, Geneva, 1994.
5. Jamet P, Ji B and the Marchoux Chemotherapy Study Group. Relapse after long-term follow up of multibacillary patients treated by WHO multidrug regimen. *Int J Lepr*, 1995; **63**: 195-201
6. Girdhar BK, Girdhar A, Kumar A. Relapses in multibacillary leprosy patients: effect of length of therapy. *Lepr Rev*, 2000; **71**: 144-153
7. Jesudasan K and others. Absence of relapse within 4 years among 34 multibacillary patients with high BIs treated for 2 years with MDT. *Int J Lepr*, 1996; **64**: 133-135
8. Vijayakumaran P and others. Fixed-duration therapy (FDT) in multibacillary leprosy: efficacy and complications. *Int J Lepr*, 1996; **64**: 123-127
9. Jesudasan K and others. Effectiveness of MDT in multibacillary leprosy. *Int J Lepr*, 1996; **64**: 128-132
10. Shaw IN and others. Long-term follow up of multibacillary leprosy patients with high BI treated with WHO/MDT regimen for a fixed duration of 2 years. *Int J Lepr*, 2000; **68**: 405-409
11. Gebre S, Saunderson P, Byass P. Relapses after fixed duration multiple drug therapy: the AMFES cohort. *Lepr Rev*, 2000; **71**: 325-331
12. Waters M. Relapse following various types of MDT therapy in MB leprosy. *Lepr Rev*, 1995; **66**: 1-9

13. Carus NH and others. Relapse of Mycobacterium leprae infection with ocular manifestations. *Clin Infect Dis*, 1995; **20**(4): 776-80
14. Croft R. Active surveillance in leprosy: how useful is it? *Lepr Rev*, 1996; **67**: 135-140
15. van Beers S, Hatta M, Klatser P. Patient contact is the major determinant in incident leprosy: implications for future control. *Int J Lepr Other Mycobact Dis,* 1999; **67**(2):119-28

Editorial comment

Vicki Krause, CDC Darwin

Back in 1991 the World Health Organisation set a goal to "Eliminate leprosy as a public health problem by the Year 2000". Well, that year has come and gone and leprosy is still with the world and elimination (defined as a prevalence of ≤ 1 case per 10 000 population – a case being one who received or required drug therapy) is still a goal for the future. A significant decline in the disease, however, has occurred since the start of short course multi drug therapy (MDT) worldwide in 1982. In countries where leprosy is still endemic, such as India and Brazil, the goal of elimination needs to be pushed back. In countries like Australia or State/Territories like the Northern Territory (NT), elimination levels have been reached. Prevalence of disease in the NT, as of June 30, 2001, is <1 per 10 000 with 1 person on active treatment and a total of 3 cases notified in the past 4 years.

Leprosy elimination is counting on many factors including that relapse following MDT is rare. Studies have shown that persisters, (i.e. viable, physiologically dormant bacilli that remain fully drug susceptible and survive for many years despite the presence of bactericidal levels of drugs) occur both in paucibacillary and multibacillary (MB) disease. Studies have shown that relapse and persisters occur at the same rate suggesting persisters may be the cause of relapse cases.* Most relapse occurs late, i.e. 6 to 9 years after treatment. As the article above suggests, following up patients in a prioritised manner is warranted, especially for MB cases.

The trick is in balancing the effort put into keeping up the expertise, awareness, research and surveillance (including appropriate active case follow-up) around leprosy with the low number of cases occurring in eg Australia while the global community moves toward global elimination. Keeping leprosy in the differential diagnosis of certain presentations is important, as is knowledge of and the capability to perform diagnostic tests (skin smears). An extensive list of those types of lesions that leprosy superficially may mimic is presented in an article in the Travel Medicine section of a recent issue of Clinical Infectious Diseases *. The possibility of leprosy needs to remain in the clinician's mind when facing these presentations. A basic principle to follow is to consider leprosy in any patient with skin or peripheral nerve lesions who resides (or did reside) in an endemic area. This especially applies to skin conditions which persist despite treatment (eg presumed fungal infections) or with idiopathic footdrop or chronic plantar ulcerations or when painless burns or injuries are noted on the hands and feet.

*Ooi W, Moschella S. Update on leprosy in Immigrants in the United States:Status in Year 2000. CID 2001:32 p.930-937

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A review of cold chain practices in the Northern Territory

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Introduction

It is well recognised that maintenance of the cold chain plays a crucial role in ensuring vaccine integrity and subsequent high levels of protection against disease.[1] Guidelines for the storage and transport of vaccines have been available and practised within the Northern Territory (NT) for the past decade.[2]

We reviewed current practices with public and private providers in both the urban and rural areas of the NT in an effort to identify if any deficiencies in cold chain management existed and to identify priorities for maintaining an efficient and viable system in the coming years. The review focused on current practice and the state of the cold chain in relation to vaccine transport and storage as well as staff knowledge.

Methods

A brief, self-administered questionnaire to determine current practices, staff awareness and knowledge of cold chain management was delivered to all known immunisation providers in the Top End of the NT including General Practitioners, Aboriginal Health Services, urban and remote community health centres (n=110). Details of participation in the Territory Health Services (THS) nationally accredited "About Giving Vaccines" course[3] was included in the survey as an indicator of staff cold chain knowledge. In addition a random sample of providers (n=35) was selected to participate in a cold chain audit of their practice. The audit consisted of tracking the temperature of a vaccine order from packing at the distribution centre through 10 consecutive days in a practice refrigerator with "Tinytag Ultra, Hastings Deering data loggers™". Pre and post audit consultations were conducted and data collated.

Results

The response rate to the survey was 61% (n=67). Fifty- percent (34) of responding services indicated they had staff performing immunisations that had not undertaken the course and 64% (22) of these had at least 2 or more staff who required training. One service indicated that training was not necessary as 'doctors were responsible for immunisation'. One person was reported as being solely responsibility for cold chain monitoring in 66% (44) of services.

Vaccine Transport

Immunisation providers in the NT order vaccines direct from their district THS hospital pharmacy where vaccines are stored in large commercial refrigerators and are monitored with automatic alarms preset at 2-8°C. From the pharmacy, vaccines are packed for transport in marked styrofoam containers/boxes with chemical Time/ Temperature indicators, freeze indicators and ice bricks. The packing is in accordance with established THS vaccine cold chain standards.

While 34% of clinics stated their staff collected vaccine orders directly from the pharmacy, 32% of vaccines were delivered by air, 18% by road and 16% by a combination of methods. Although most transportation of vaccines by air took less than 6 hours, delays or diversions to other communities and the inadvertent leaving of vaccines on tarmacs did occur with a small number of the deliveries. Similarly, drive times to clinics varied greatly from less than 30 minutes in the urban area to greater than 6 hours in the remote areas. Overall, 11 different commercial couriers were identified and delivery to remote areas could involve several couriers for a single batch of vaccine.

Vaccine Storage

Refrigerators dedicated to vaccine storage were reported by 91% of vaccine providers. However, in many general practices where refrigerator space was limited, other pharmaceuticals (i.e. insulin, eye drops, emergency drugs) were stored in the refrigerator with the vaccines in accordance with The Australian General Practice Accreditation Limited (AGPAL) standards. All clinics had refrigerator thermometers (80% being electronic

maximum/minimum models) and 93% of clinics recorded daily temperatures in accordance with THS and the National Health and Medical Research Council (NHMRC) guidelines.[1]

Electronic data from the loggers indicated that all the fridges maintained temperatures between 2°-8°C for the study period. Freezing was not detected in any vaccine storage units. Maximum temperatures of 21°C were recorded in 2 refrigerators in rural communities but were associated with power failures and were short term (less than 8 hours). Temperatures during transport were often higher with a range of between 10°C-15°C. During transport and storage, all vaccines were accompanied by Freeze indicators and Time/Temperature monitors. Thirty one percent of participants indicated checking these monitors daily, 45% on a weekly basis and 23% when vaccines were used.

Forty three percent of providers indicated that their vaccine refrigerators do not have the capacity to accommodate additional vaccines.

Discussion

Ensuring the integrity of the cold chain across more than 120 isolated sites in the NT is a significant logistical challenge. The effects of distance, high staff turnover and diverse health service infrastructures compound an already difficult problem. Given the vastness of the Territory, isolated locations of many vaccine providers and seasonal change in access to many locations, a variety of transport methods are necessary to facilitate vaccine delivery. Even with these barriers delays were minimal and all the vaccines arrived well within cold chain parameters.

Vaccine distribution in the Top End is reasonably complex, with multiple transport modes and routes and as such, there is room for error. Several states have now centralised vaccine distribution systems, which provide greater control over vaccine supply and accountability. The authors agree that centralising vaccine distribution and reducing the number of couriers utilised in vaccine delivery would minimise risk of breaks in the cold chain. However it is difficult due to the reasons stated and may not be cost effective. The concept should however be further investigated as a long-term goal for NT immunisation programs.

It was extremely reassuring that none of the vaccines were exposed to freezing during transport or storage. Historically, freezing during transport and particularly storage has been as high as 48%[4]. Continued monitoring of freeze sensitive vaccines remains a high priority and is an essential management tool. The 10°C-15°C temperatures recorded during transport were of short duration and are not an area of concern.

This review revealed a broad awareness of the importance and need to maintain diligence in cold chain practices in the NT. It highlighted the need for ongoing education and monitoring of cold chain management. It also suggests a need to streamline vaccine delivery to reduce the risks of vaccine wastage. The review follows current recommendations to move toward the purchase of vaccine dedicated refrigerators for storage and the need to continuously provide opportunity for staff training in vaccine management.

In this, a year that foreshadows the introduction of new vaccines and further emphasis on adult immunisation programs, the need for more vaccine storage space will inevitably be required. Specialised vaccine refrigerators, in which modification is not required and all of the refrigerator space is available for vaccine storage, may offer a solution to the need for more vaccine storage space. It is realised that these refrigerators are nearly double the cost of a domestic refrigerator but the advantages of regulated temperature control and increased storage space need to be considered. Providers upgrading their vaccine storage refrigerators should be encouraged to investigate this option*.

Although comparison of cold chain management/practices in relation to completion of the course has not been undertaken it would be useful to determine if a positive link exists. The THS vaccine course is flexible and relatively inexpensive. Vaccine management practitioners should be encouraged to participate. Regular updates should also be encouraged and mechanisms put in place to facilitate this process.

*Contact Nan Miller on 8922 8564 or nan.miller@nt.gov.au for detailed information regarding specialised vaccine storage refrigerators and Workforce Development on 8922 8715 for information on the THS vaccine course.

References

1. National Health & Medical Research Council. The Australian Immunisation Handbook. Canberra: Australian Government Publishing Service, 2000.
2. Miller, N. C. The Vaccine Cold Chain: Guidelines for the safe transport and storage of vaccines. 1994. Darwin, Centre for Disease Control, Territory Health Services.
3. Centre for Disease Control. About Giving Vaccines: an accredited short course for vaccine providers. 1998. Darwin, Territory Health Services.
4. Miller NC & Harris MF. Are childhood immunisation programs in Australia at risk,: Investigation of the cold chain in the Northern Territory. *WHO Bulletin* 1994, 72;3:401-408.

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Case history: Neurological disease in a cat

Helen Parkes, Berrimah Veterinary Laboratories

An 18 month old, desexed male cat died after several days of showing unusual neurological signs. It was submitted to BVL for post-mortem examination because Australian bat lyssavirus had been suggested as a possible differential diagnosis for its condition. The cat had been presented to the vet clinic with onset of staggering over the course of one day, and left foreleg lameness. The cat was bright, but had its third eyelids protruding. It had recent probable cat fight wounds evident. The cat had arrived from Queensland 3 weeks before, so a paralysis tick was a possibility, but none was found. Over the next 4 to 5 days, it had a fluctuating temperature, muscle trembling and developed left foreleg paralysis and right foreleg paresis. The cat was still bright and continued to eat, although in the last 24 hours before it died it deteriorated dramatically, becoming unable to lift its head. Its breathing was abdominal, but did not appear to be laboured. What seemed unusual was that the "front end" of the cat was most affected by the condition, while the hindlegs appeared unaffected. The cat was treated with antibiotics, anti-inflammatory drugs and fluids, but died 5 days after presentation.

Post-mortem examination revealed an abscess of the medial toe of the left hindfoot, discharging pus from around the claw. There was a single, small (1-2mm diameter) abscess on the spleen and several similar abscesses under the capsule of the liver. The lungs had patchy dark red discolouration. The meninges were slightly dark and cloudy.

Swabs of the toe and meninges, and samples of fresh liver and spleen all grew *Burkholderia pseudomallei* (the causative agent of melioidosis). Histological examination showed typical acute "melio" abscesses in the spleen and liver. The cervical and thoracic spinal cord and the brain stem showed severe, focal necrosis with purulent inflammation. Necrosis and inflammation extended into some of the spinal nerve roots. The spinal meninges also showed extensive purulent inflammation.

This was the first time we have seen such a convincing case of *Burkholderia pseudomallei* septicaemia and meningoencephalitis with spinal cord involvement in a cat. A nice explanation for the case would be that this cat (being new to the Top End) had no previous contact with melio (hence no protective antibodies) and became infected with the organism following a cat fight. Spinal cord necrosis and abscessation have been seen in other species with *B. pseudomallei* infection.

Editorial comment

Bart Currie, Menzies School of Health Research and NT Clinical School

This is a very well documented case of neurological melioidosis affecting a cat. Many features are similar to neurological melioidosis in humans, including the presentation with limb paralysis and the presence of abscesses in internal organs but a more diffuse inflammatory process affecting brainstem, spinal cord and extending to spinal nerve roots. Dr Jodie Low Choy who is currently at the Territory Wildlife Park has recently published an excellent summary of animal melioidosis in Australia, drawing on the extensive NT experience of BVL and other veterinary colleagues. Neurological melioidosis is well documented in horses, sheep and goats, along with other diverse clinical melioidosis presentations as seen in humans. Pigs in the NT are usually asymptomatic and melioidosis is often only diagnosed in pigs when abscesses in internal organs are found on slaughtering. Melioidosis is uncommon in cats and dogs, although one well known RDH physician heroically and successfully adapted the RDH melioidosis intensive therapy protocol for his pampered pooch.

The issue of whether there can be acquired immunity to melioidosis from repeated environmental exposure to the bacteria *Burkholderia pseudomallei* in endemic regions such as the Top End remains to be resolved. If this does occur, as hinted at by this report and some epidemiological data from Thailand, it suggests current attempts at effective vaccine development may well be successful.

References

1. Low Choy J, Mayo M, Janmaat A, Currie BJ. Animal melioidosis in Australia. *Acta Tropica* 2000;74:153-158.
2. Currie BJ, Fisher D, Howard D, Burrow JNC. Neurological melioidosis. *Acta Tropica* 2000;74:145-151.

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BreastScreen NT

Beth Amega, Policy/Promotions Officer, Womens Cancer Prevention Program

Breast cancer is a major health issue for all women. The lifetime risk of Australian women developing breast cancer before age 75 years is 1 in 11.[1] During the 10 year period from 1987 to 1997, breast cancer rates were lower in the female Northern Territory (NT) population than in other states. In this period 376 women developed breast cancer (342 NT non-Aboriginal, 34 Aboriginal) and there were 61 deaths from breast cancer (50 non-Aboriginal and 11 Aboriginal women).

Despite significant research there is no clear answer as to the cause of breast cancer and therefore no means of prevention. We do know that breast cancer is a disease of aging, over 70% of breast cancers are found in women over 50 years of age. Family history is not a strong risk factor as 8 out of 10 women with breast cancer have no family history of the disease. The only known risk factor for breast cancer is "being a woman and getting older".

Evidence based on trials and research in the 1960's supports large scale population-based screening programs utilising mammography as the primary screening tool. The National Program for the Early Detection of Breast Cancer, a Commonwealth initiative, commenced in 1991 and later changed it's name to BreastScreen Australia. BreastScreen NT, the NT arm of the National Program, commenced operation in Darwin with a full time Screening and Assessment Service in November 1994. A part time service commenced in Alice Springs in January 1996. A part time service also operates in Tennant Creek, Katherine and Nhulunbuy with a relocatable machine due to the low number of eligible women in these areas.

The aim of the program is to reduce the number of deaths from breast cancer. To achieve this requires a large percentage of the eligible population to participate and for optimal screening quality to detect lesions prior to development of any visible signs or symptoms. Early detection means greater treatment options and the potential for complete cure.

The national benchmark for an effective program is 70% participation rate of the target population, women aged 50–69 years, and a 75% re-screen rate. Promotional activities are the key to recruiting women to the program and these are targeted at women aged 50-69 years. These women are actively encouraged to participate in screening every 2 years. Women aged 40-49 years and women over 70 years are not within the main target group as defined by the National Program but are welcome to access screening if they so request. Women under 40 are excluded from the program, as there is no evidence of benefit from mammography screening in this age group.

Since becoming operational in Darwin in November 1994, recruitment strategies have focused on increasing awareness of the service throughout the community and among health professionals. This has proved successful with a high rate of awareness of the service and its purpose. Strategies are currently aimed to target those women who are hard to reach such as Aboriginal women, culturally and linguistically diverse women (CALD) and women in lower socioeconomic groups. The Women's Cancer Prevention Program employs project officers specifically to work with indigenous and CALD women. Two major promotional events held each year specifically targeted at women in the Darwin district from these areas include the Indigenous Women's Health Expo held on 20 June 2001 and the Women's Health Day for CALD women to be held on 28 August 2001. October 2001 is breast cancer awareness month and will be used to target women NT- wide especially in the special needs groups.

Current screening rates for NT services

During the 2 year period ending 31 December 2000, 65.9% of all eligible NT women aged 50-69 years had attended BreastScreen NT. As shown in the graph below, the yearly screening rate appears to be falling. This is of concern to the program although can in part be attributed to the itinerant nature of the NT population.

Figure 1 BSNT recruitment 50-69 years 1998-2000

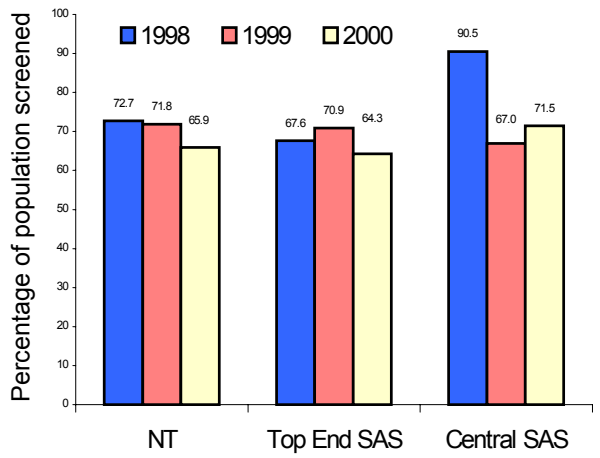

Table 1 BSNT recruitment 50-69 years 1998-2000

	NT			Top End SAS			Central SAS		
	Pop.	No. screened	Rate (%)	Pop.	No. screened	Rate (%)	Pop.	No. screened	Rate (%)
1998	3323	2415	72.7	2589	1751	67.6	734	664	90.5
1999	3824	2746	71.8	3043	2156	70.9	881	590	67.0
2000	4194	2765	65.9	3230	2076	64.3	964	689	71.5

Table 2 BSNT recruitment and malignancies 1998-2000

	Number screened	Number recalled to assessment	Invasive malignancies found
1998	3723	133	19
1999	4076	113	13
*2000	4150	98	15

*Data for 2000 may be incomplete

Assessment

BreastScreen NT provides a full assessment service for any woman found to have a screen detected abnormality.

In Darwin and Alice Springs:

- 4.2% of women in their first round of screening in the year 2000 were recalled for assessment,
- 2.0% of women in their subsequent rounds of screening in the year 2000 were recalled for assessment.

Of those called for assessment only a small number are found to have a malignancy.

Summary

To continue to be an effective public health measure, the 70% benchmark targeted participation rate must be met. Having obtained this in the past the fall off in year 2000 needs to be fully evaluated.

If you wish to comment on this article or make other comments about BreastScreen NT please contact Karen Finch by email or phone 8922 5500.

References

1. Australian Institute of Health and Welfare. Cancer in Australia 1997.

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NT Malaria notifications – January to March 2001

Merv Fairley, CDC, Darwin

Sixteen notifications of malaria were received for the first quarter of 2001. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

Number of cases	Origin of infection	Reason exposed	Agent	Chemoprophylaxis	Comments
3	PNG	Work	P.vivax	no	diagnosed ASH
1	PNG	Student	P vivax	no	diagnosed RDH
2	Indonesia	Holiday	P vivax	no	diagnosed RDH
1	Indonesia	Holiday	P vivax	yes	diagnosed RDH
1	Indonesia	resident	P vivax	no	diagnosed RDH
1	Indonesia	resident	P falc	no	diagnosed RDH
3	East Timor	work	P vivax	yes	diagnosed Westerns
1	East Timor	work	P vivax	no	diagnosed RDH
2	East Timor	work	P vivax	yes	diagnosed QML
1	East Timor	work	P vivax	yes	diagnosed RDH

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Points to note regarding notifications on page 36

- The reason for an increase in reporting of cryptosporidiosis in 2001 is unclear. There is little evidence of any links among most cases. Given that cryptosporidiosis has only been notifiable for a couple of years, these figures may reflect the 'true' background rate.

- Ross River Virus numbers are influenced by the climate and numbers were high in the Barkly region because of record rainfall in 2001.

- Rotavirus numbers are much less than for the same period last year noting that East Arnhem had an outbreak in 2000 with 40 cases within a 5 week period. (however, see **Grab This**, page 38)

- There has been an increase in the number of trachoma (*Chlamydia trachomatis*) notifications from an Aboriginal community in Darwin Rural which has embarked on a screening and eradication program.

- Kunjin, Kokobera, Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Gastroenteritis, Haemolytic Uraemic Syndrome, Hepatitis C (incidence), Hepatitis D & E, Hydatid Disease, Leprosy, Lymphogranuloma venereum, Measles, Ornithosis, Poliomyelitis, Typhus, Vibrio Food Poisoning, Viral Haemorrhagic Fever and Yersiniosis are all notifiable but had "0" notifications in this period.

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Disease Control Staff Updates

Deidre Ballinger has recently been appointed as AIDs/STD project officer in Darwin CDC. Deidre has been a midwife in the NT for over 5 years, holding positions in both rural and urban settings. She has most recently been working in a community health.

NT notifications of diseases by districts
1 January to 31 March 2001 and 2000

Diseases	Alice Springs		Barkly		Darwin		East Arnhem		Katherine		Total	
	2001	2000	2001	2000	2001	2000	2001	2000	2001	2000	2001	2000
Acute Rheumatic Fever	5	0	0	0	0	0	0	0	1	0	6	0
Adverse Vaccine React.	1	0	1	0	4	8	2	0	0	0	8	8
Amoebiases	1	0	0	0	0	0	0	0	1	0	2	0
Arbovirus infections												
Murray Valley Encephalitis	2	0	0	0	0	0	0	0	0	0	2	0
Barmah Forest Virus	0	0	4	0	8	1	4	0	5	1	21	2
Dengue	0	0	0	0	14	63	0	0	0	0	14	63
Ross River Virus	7	0	62	5	81	63	3	6	40	20	193	94
Atypical Mycobacteria	0	0	0	0	1	0	0	0	0	0	1	0
Campylobacter	21	19	5	2	36	20	5	1	5	10	72	52
Chlamydia	100	98	2	7	111	71	31	17	23	19	267	212
Chlamydia Conjunct.	0	0	0	0	35	12	0	1	3	1	38	14
Congenital Syphilis	0	0	0	0	0	0	0	0	1	0	1	0
Cryptosporidiosis	34	41	3	3	44	15	6	1	28	6	115	66
Diphtheria	0	0	0	0	1	0	0	0	0	0	1	0
Donovanosis	1	2	0	0	0	0	0	0	0	2	1	4
Glomerulonephritis	2	0	0	0	4	4	1	0	0	0	7	4
Gonococcal Disease	160	151	5	14	62	33	32	15	32	29	291	242
Gonococcal Conjunct.	0	4	0	0	0	0	0	0	0	0	0	4
Gon Opthalmic Neonatal	1	0	0	0	0	0	0	0	0	0	1	0
Haemophilus Inf all type	1	0	0	0	0	0	0	0	0	0	1	0
Hepatitis A	1	7	0	0	4	13	1	0	0	4	6	24
Hepatitis B	1	0	0	1	0	1	0	0	1	2	2	4
Hepatitis C (prevalence)	10	2	0	1	46	23	4	0	2	2	62	28
HIV infections	0	0	0	0	3	2	0	0	0	0	3	2
HTLV-1	12	4	0	0	0	0	0	0	0	1	12	5
Influenza	3	0	0	0	1	2	0	6	2	2	6	10
Legionnaires Disease	0	0	0	0	1	0	0	0	0	0	1	0
Leptospirosis	0	0	0	0	0	0	0	0	1	0	1	0
Listeriosis	0	3	0	0	0	0	0	0	0	0	0	3
Malaria	1	2	0	0	10	11	0	0	0	0	11	13
Melioidosis	0	2	1	0	12	15	3	1	3	4	19	22
Meningococcal Infection	2	2	0	0	0	0	0	0	0	0	2	2
Mumps	0	0	0	0	0	1	0	0	0	0	0	1
Pertussis	3	2	0	0	2	0	0	0	2	0	7	2
Pneumococcal Disease	9	9	2	0	5	0	0	0	2	2	18	11
Rotavirus	7	10	2	6	0	18	0	44	5	5	14	83
Rubella	0	0	0	0	0	0	0	1	0	0	0	1
Salmonella	30	31	4	4	75	50	10	6	20	21	139	112
Shigella	20	12	2	0	13	5	3	8	1	1	39	26
Syphilis	0	20	3	2	15	20	14	2	11	12	43	56
Trichomonas	61	95	14	6	57	52	48	79	56	61	236	293
Tuberculosis	1	3	0	0	6	12	0	0	0	2	7	17
Typhoid	0	0	0	0	1	0	0	0	0	0	1	0
Total	**497**	**519**	**110**	**51**	**652**	**515**	**167**	**188**	**245**	**207**	**1671**	**1480**

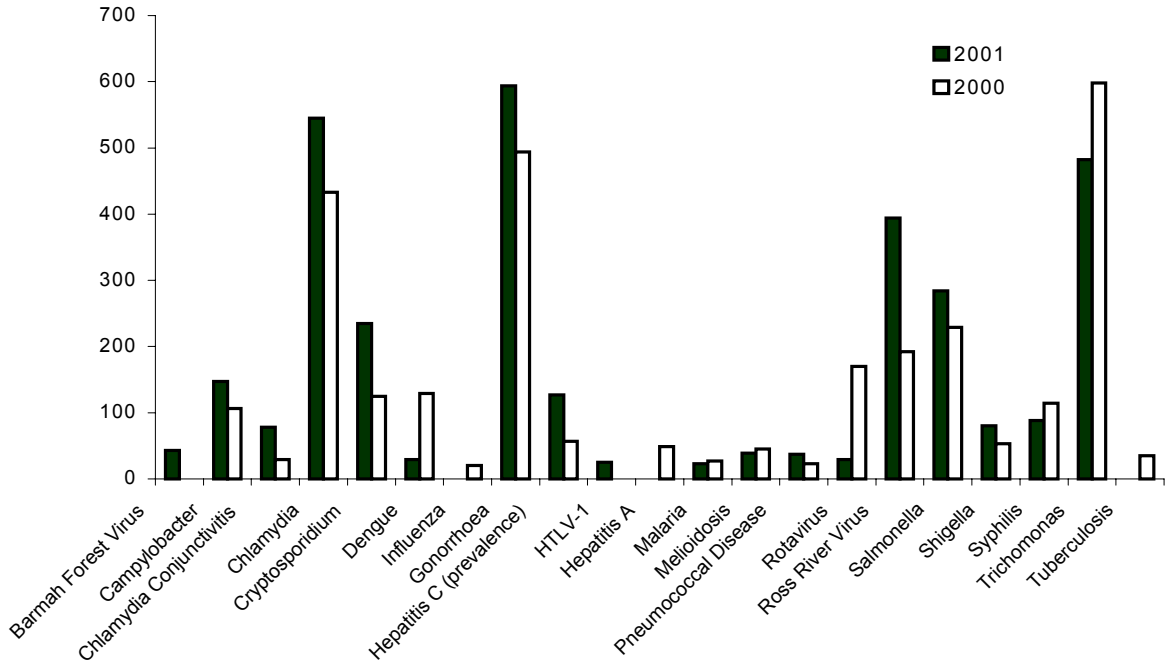
Notification of Infectious Diseases - Telephone: 8922 8044 Fax: 8922 8310

Notified cases of vaccine preventable diseases in the NT by report date 1 January to 31 March 2001 and 2000

DISEASES	TOTAL		No. cases among children aged 0-5 years	
	2001	2000	2001	2000
Congenital rubella syndrome	0	0	0	0
Diphtheria	1	0	0	0
Haemophilus influenzae type b	0	0	0	0
Hepatitis B	2	4	0	0
Measles	0	0	0	0
Mumps	0	1	0	0
Pertussis	7	2	1	1
Poliomyelitis, paralytic	0	0	0	0
Rubella	0	1	0	1
Tetanus	0	0	0	0

- Mumps is largely under-reported.

NT wide notifiable diseases 1 January to 31 March 2001 and 2000



Rates <10/100,000 not listed
NT est resid. Pop-195,905 supplied by Epidemiology & Statistical Branch, THS

Grab this!

Communicable Disease Notes

Pertussis

So far 38 cases have been notified in 2001 compared to 6 for the whole of 2000. Only 9 cases were under 10 years of age and 14 were aged 10-14 years. Almost all cases have been vaccinated. Cases have occurred throughout the Territory. Most cases appear unlinked although there is some suggestion of spread within one primary school and there was some 'relatedness in the East Arnhem cases'. Given the need for effective contact tracing and the offering of appropriate prophylaxis, especially to protect infants, early notification on clinical suspicion is encouraged.

Rotavirus

Rotavirus gastroenteritis has caused significant morbidity so far this year. The 'outbreak' total to date is 339 cases and began in the Alice Springs region in late April / early May which has accounted for 53% (n=179) of the notifications. Katherine followed, and now has 20% (n=68) and then Darwin, now with 22% (n=76) of the notifications. The first 2 notifications from East Arnhem have just been received. The epidemics in the Centre and in Katherine district are subsiding. Just over half of the Alice Springs cases are serotype G9; samples from the Top End have yet to be serotyped. Dr Alex Brown from Alice Springs comments that "The dynamics of this outbreak has made it particularly difficult to delineate the exact source of infection, and the transmission cycles that have led to the vast number of cases (with attack rates of confirmed rotavirus representing 11% of all children under two years of age in the region). Very few of the cases had any definitive contact with known rotavirus positive cases, or with family members with symptoms of gastroenteritis. Although there was evidence of geographical links between some cases with similar serotypes, overall, the temporal and geographical patterns do not fit with a common source outbreak, or a single epicentre with secondary and tertiary cases. The rapid onset of rotavirus activity across vast distances with mixed rotavirus serotypes is difficult to adequately explain."

Meningococcal Infection

There have been 7 cases notified this year; 6 group B and 1 group C. Four of the cases were from Darwin and all occurred over a 3 week period. All 4 cases were from Aboriginal communities with epidemiological links between 3 of them. Extensive contact tracing was performed and prophylaxis given. The health staff at these communities acted promptly and very efficiently to treat all of the listed contacts in a timely fashion and to educate their communities. Fortunately, all 4 cases have made good recoveries. The remaining 3 cases (2 from the Centre) appear to be sporadic cases.

Murray Valley Encephalitis and Kunjin Virus

Earlier this year there were 2 cases of Murray Valley Encephalitis (MVE) notified –both from the Alice Springs region. Additionally, a German tourist was diagnosed with MVE on returning to Germany, after having acquired the disease while travelling in the Northern Territory (NT). A man was also diagnosed in Western Australia having acquired the disease most likely in the NT. Last year there was a total of 7 notified cases of MVE and prior to that the last cases were reported in 1993.

There have also been 2 cases of Kunjin Virus notified this year, again from the Alice Springs region with the most recent case notified in May. Additionally, also in May, an NT tourist returned to his home in Melbourne and was diagnosed with Kunjin Virus encephalitis.

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Conjugate pneumococcal vaccine now available in the NT !

On June 1, 2001 the first doses of Prevenar ® (Wyeth-Lederle), the only licensed conjugate pneumococcal vaccine presently available in Australia were administered to eligible 2 month olds in the Northern Territory (NT). This vaccine has long been awaited as a strategy to assist in the prevention of invasive pneumococcal disease (IPD) which causes mainly pneumonia, septicemia and meningitis. The vaccine protects against 7 different *Streptococcus pneumoniae* (the organism responsible for IPD) serotypes (4, 6B, 9V, 14, 18C, 19F, 23F) and can be used in infants from 6 weeks of age – thereby providing coverage for the age group most 'at risk' for IPD – those < 2 years. While there are about 100 different pneumococcal serotypes the 7 present in Prevenar® account for about 58% and 72%, respectively, of those found in IPD cases in the < 2 year old Aboriginal and non-Aboriginal children.

A National Childhood Pneumococcal Vaccination Program to protect babies and children at high risk from pneumococcal disease started throughout Australia on July 1 2001 and so - - - - the NT got a head start.

Those eligible for Prevenar® in the NT are:

in Central Australia -

- Aboriginal children < 5 years of age
- Non-Aboriginal children < 2 years of age

in the Top End -

- Aboriginal children < 2 years of age

Territory-wide -

- all children with medical risk factors which predispose them to high rates of severe IPD eg those with impaired immunity ie haemoglobinopathies, congenital immune deficiencies, asplenia, HIV infection, nephrotic syndrome, or those with anatomical abnormalities such as congenital cyanotic heart disease and cerebral spinal fluid leaks.

The eligibility for this free vaccine is based on the burden of IPD. In the < 2 year old population Central Australian Aboriginal children have a rate of 1534 per 100 000, one of the highest reported rates in the world. The rate in Top End Aboriginal children, 326 per 100 000, is also high at and Central Australian non-Aboriginal children have a rate of 218 per 100 000. Non-Aboriginal Top End children < 2 years have much lower rates (~80 per 100 000), similar to non-Aboriginal children in other areas of Australia.

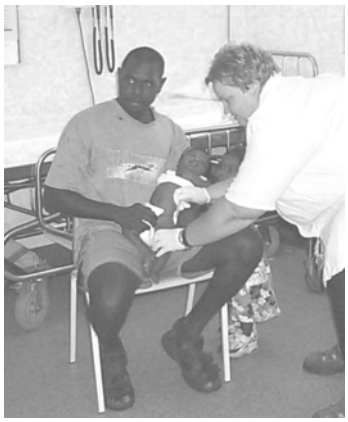
Prevenar® was introduced into the NT schedule on 1 June 2001 for those eligible children born on or after 1 April 2001 which includes all Aboriginal children and other 'at risk children'(Central Australian non Aboriginal children and children with medical risk factors that predispose them to high rates or severity of IPD).

The 23-valent polysaccharide pneumococcal vaccine (Pneumovax®23) will be given as a single dose to all Aboriginal infants at 18 months of age. Central Australian Aboriginal children < 5 years of age that have received the 23 valent polysaccharide vaccine already will be given one dose of Prevenar® during the catch-up program.

In the new NT schedule all 'at-risk' children will be eligible for Prevenar at 2, 4 and 6 months and Aboriginal children will receive an additional 23-valent polysaccharide vaccine at 18 months.

PowerPoint presentations on the reasons for, and introduction of, the 7-valent conjugate pneumococcal vaccine (Prevenar®) are available from local NT CDC units. *The Medical Journal of Australia* produced a full supplement on "Pneumococcal disease in Australia" on 2 October 2000 which includes the article "Invasive pneumococcal disease in the Northern Territory of Australia , 1994-1998".

Prevenar® being given at Bagot Clinic 1 June 2001.



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NT Pneumococcal Vaccine Childhood Catch-up Schedule

Central Australia

	Age at first dose	Primary series *(7vPCV)	Additional doses **(23vPPV)
Aboriginal	3-6 months	3 doses 2 months apart	18 months
	7-17 months	2 doses 2 months apart	18 months or 2 months after primary
	18-23 months	1 dose	2 months after primary
	24-59 months	1 dose	2 months after primary
Non-Aboriginal	3-6 months	3 doses 2 months apart	None
	7-17 months	2 doses 2 months apart	None
	18-23 months	1 dose	None

***7vPCV** – 7-valent conjugate pneumococcal vaccine (Prevenar®)
****23vPPV** – 23-valent polysaccharide pneumococcal vaccine (Pneumovax®23)

Note: Central Australian Aboriginal children who have received 1 dose of polysaccharide pneumococcal vaccine between 2-5 years of age should have a single dose of conjugate pneumococcal vaccine, only.

Top End

	Age at first dose	Primary series *(7vPCV)	Additional doses **(23vPPV)
Aboriginal	3-6 months	3 doses 2 months apart	18 months
	7-17 months	2 doses 2 months apart	18 months or 2 months after primary
	18-23 months	1 dose	2 months after primary

***7vPCV** – 7-valent conjugate pneumococcal vaccine (Prevenar®)
****23vPPV** – 23-valent polysaccharide pneumococcal vaccine (Pneumovax®23)

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