Surveillance of invasive meningococcal disease in Queensland, 2002

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Abstract

During 2002, 124 cases of invasive meningococcal disease were notified in Queensland. This was similar to the previous year (n=128). Four (3.2%) of the cases died. Trends by age and serogroup were generally similar to previous years and were consistent with the overall patterns of this disease in Australia. However, an apparent increase in serogroup C, which infected 41 per cent of cases, needs continued monitoring. This report highlights the need for continued surveillance of morbidity and mortality patterns and management of this disease. Ongoing surveillance will monitor the impact of the National Meningococcal C Vaccination Programme, commenced in early 2003. This report also highlights the need for ongoing community education to ensure people seek medical attention early after onset of the illness. This report shows that when general practitioners considered meningococcal disease as a diagnosis, their patients were admitted to hospital sooner than patients in whom this diagnosis was not initially considered. Acknowledging that early disease may present diagnostic difficulties, further awareness raising amongst general practitioners is required to promote early recognition and referral. *Commun Dis Intell* 2003;27:342–351.

Keywords: invasive meningococcal disease, communicable diseases, surveillance

Introduction

Invasive meningococcal disease (IMD) is notifiable to Queensland Health by laboratories identifying a laboratory confirmed case of disease and also by clinicians, on clinical suspicion of disease. The data are maintained on the Notifiable Conditions database (NOCS), and have been collated since 1993. In 1999, enhanced surveillance for invasive meningococcal disease was established throughout Queensland. Communicable diseases staff of the Public Health Units coordinate public health responses to notified cases and conduct enhanced surveillance. Queensland Health reported on enhanced surveillance for the years 1999,¹ 2000,² 2001³ and 2002.⁴ This paper is derived from the 2002 report.

The purposes of this paper are:

- to describe the epidemiology of invasive meningococcal disease in Queensland in 2002;
- to describe risk factors for dying of IMD identified in the four year period since enhanced surveillance began;
- to describe trends of the disease since 1993; and

 to discuss the implications of these findings for ongoing surveillance and control of this disease with particular reference to the introduction of vaccination against *Neisseria meningitidis*.

Methods

The following definitions for confirmed and probable cases of invasive meningococcal disease were used:

Confirmed cases of invasive meningococcal disease were defined as: a clinically compatible illness with at least one of the following—isolation of *Neisseria meningitidis* from an otherwise sterile body site, *or* detection of gram-negative intracellular diplococci in cerebrospinal fluid (CSF) or petechiae, *or* a positive polymerase chain reaction (PCR) test on CSF, blood or serum, *or* a positive meningococcal antigen test on CSF, *or* detection of meningococcal IgM in serum.

The PCR test has been used in Queensland from 1999 onwards, and the IgM test was introduced in 2000.

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Probable cases were defined as: a clinically compatible illness with at least one of the following—a petechial or purpuric rash, *or* isolation of *N. meningitidis* from a throat swab *or* close contact with a confirmed case.

Meningococcal conjunctivitis as diagnosed by isolation of *N. meningitidis* from the conjunctiva of a patient with conjunctivitis is not strictly speaking an invasive disease but is included in surveillance because it may be associated with invasive disease in the patient or with invasive meningococcal disease in a contact.⁵ These cases will be reported below but not included in analyses of invasive meningococcal disease.

Public health units seek information on each notified case from the attending medical staff and from the patient or next of kin. A standardised case reporting form is used (Appendix 1). Information on previous years was taken from other published reports and from the database; variation from data in previous reports occurs due to data cleaning and obtaining additional information for the dynamic database. Analysis was performed in Excel, EpiInfo 6⁶ and Stata.⁷ Chi-square, Yates corrected or Fisher's exact tests were used where appropriate.

Results

There were 124 cases of invasive meningococcal disease in Queensland in 2002. This represented an incidence of 3.4 cases per 100,000 population, which is similar to 2001 and higher than the years between 1993 and 2000 when it ranged between 1.9 and 3.2 cases per 100,000 population (Figure 1). There were also three cases of meningococcal conjunctivitis. These cases are excluded from further analysis of the invasive meningococcal cases in 2002.

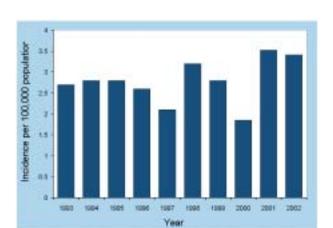


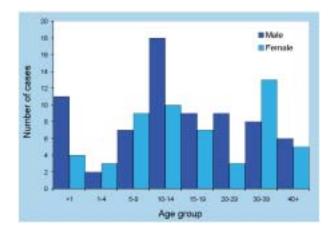
Figure 1. Annual incidence per 100,000 population of invasive meningococcal disease, Queensland, 1993 to 2002

Of the 124 invasive meningococcal cases, 118 (95%) were laboratory-confirmed and 6 (5%) were probable cases. There were four deaths in 2002, representing a case fatality rate of 3.2 per cent. This is not significantly different from the case fatality rate in 2001 of 8.7 per cent (p=0.07).

Age and gender distribution

In Queensland during 2002, 33.9 per cent of all cases of invasive meningococcal disease occurred in children below 10 years of age; 25.0 per cent of all cases were under five years of age; 12.1 per cent were under one year and 12.9 per cent were aged 1–4 years. Persons aged 15–29 years accounted for 35.5 per cent of cases. Of the 124 cases, 70 were males (56%) and 54 were females (44%) (Figure 2).

Figure 2. Number of invasive meningococcal cases, Queensland, 2002, by age group



As in previous years, the rate was highest among infants. The 15–19 year age group had the second highest rate in 2002. The rate for this age group has risen steadily over the last three years such that the rate in the 15–19 year age group in 2002 was significantly higher than the rate for that age group in 2000 (p<0.05). In contrast, rates amongst the 1–4 year age group in 2002 were lower than in 2001 and were the lowest for 10 years for this age group, although the difference was not statistically significant (p>0.05) (Table 1).

Indigenous status

In 2002, Indigenous status was identified for all cases of invasive meningococcal disease. Of the 124 cases, six (5.5%) were recorded as Indigenous. This is not significantly different from the Queensland population where 3.1 per cent are estimated to be Indigenous (p=0.2). Three of the six Indigenous cases were below five years of age; one was under one year. Of the 31 cases under five years, 9.7 per cent were Indigenous; this also is not significantly different from this age group in Queensland, where 6.2 per cent are estimated to be Indigenous (p=0.6).

Table 1.	Age-specific annual incidence of invasive meningococcal disease, Queensland, 1993 to 2002,
	(age-specific rates per 100,000 population*)

Age group (years)	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002
<1	37.4	30.8	28.6	24.2	24.2	33.0	24.2	15.0	28.2	30.2
1–4	16.0	10.3	11.8	10.3	13.4	12.9	10.8	8.2	12.0	8.0
5–9	2.1	1.2	1.7	4.6	0.4	3.3	4.6	1.6	6.1	4.2
10–14	3.2	2.8	1.2	2.0	2.8	2.8	1.2	1.9	2.3	1.9
15–19	4.6	9.3	8.8	7.6	5.5	8.4	6.7	5.3	8.7	10.6
20–29	1.8	2.4	2.8	2.4	1.4	2.6	2.8	1.9	4.9	3.1
30–39	0.6	0.6	1.2	0.4	0.4	0.8	0.4	0.4	0.7	2.2
40+	0.5	1.0	0.9	0.7	0.4	1.2	1.1	0.5	1.0	1.4
Total	2.7	2.8	2.8	2.6	2.1	3.2	2.8	1.9	3.5	3.4

* Rates calculated using 1996 census data for 1993 to 2000, rates for 2001 and 2002 calculated from 2001 estimated resident population

Seasonal variation

As in previous years, the incidence of disease peaked during winter or early spring. Fifty per cent of cases occurred in the four months June to September with August recording the highest number of notifications (23,18.5%).

Clinical presentation

Thirty-five cases (28.2%) had meningitis alone on clinical presentation and 59 (47.6%) presented with septicaemia alone. Sixteen patients (12.9%) had both meningitis and septicaemia (Table 2). This was not significantly different from the disease presentation profile in 2001^3 or 2000^2 (p 0.3). Overall, 82 (66%) of the 124 cases developed a rash during the reporting period; however, 45 (76%) cases with septicaemia presented with a rash (Table 2).

Risk factors

Links with other cases

Of the 124 cases, there was one cluster of epidemiologically and microbiologically linked cases. Two males and two females aged between 19 and 40 years from a central Queensland town and surrounding area presented with invasive meningococcal disease within 37 days in July/August 2002. All four had a good clinical outcome. Three cases were identified as serogroup C and one as a serogroup Y. Not all isolates could be typed, however, the serogroup Y and one of the serogroup C isolates had the same sero-subtype (P1.5) and it was postulated that the serogroup Y may have undergone a capsule change. These cases met the national guideline criteria of a cluster and in addition to usual public health responses, a vaccination program was implemented. Between 20 September and 23 September, 2,299 men and women in the risk age group of 18-40 years, who lived or worked in a 15 kilometre radius of this town since 1 July 2002, were vaccinated. A

Table 2.	Clinical	presentation	of invasive	meningococcal	disease,	Queensland, 2002

Clinical presentation	Number with a rash	Number without a rash	Total number	%
Meningitis	19	16	35	28.2
Septicaemia	45	14	59	47.6
Meningitis + septicaemia	11	5	16	12.9
Septic arthritis	1	3	4	3.2
Eye disease (intraocular)	0	1	1	0.8
Not stated	0	9	9	7.3
Total	82	42	124	100

polysaccharide (serogroups ACW135Y) vaccine was used until stocks were exhausted and the remainder were vaccinated with a conjugate (serogroup C) vaccine. No other cases were detected in this area in 2002.

Child care

Eight sporadic cases had associations with child care centres; six were in the 0–4 age group, one was aged 9 years in after school care and the other was an 18-year-old child care attendant.

Laboratory diagnosis

Of the 118 laboratory-confirmed cases, 94 (80%) cases were diagnosed by isolation of *N. meningitidis* from a clinical specimen and at least one other test. Only 23 (19%) of the 118 were diagnosed by culture alone. Although a total of 51 cases overall had meningococcal DNA detected, 16 of these cases were diagnosed by nucleic acid tests alone, reflecting the benefits to surveillance of these advanced tests. Twenty-six (21%) of the cases were detected using nucleic acid tests; the diagnoses of only three of these cases did not include nucleic testing. A total of 68 cases overall had positive microscopy; no cases were diagnosed by microscopy alone.

Serogroups, serotypes and serosubtypes

Of 124 cases, 117 isolates or DNA samples (94.4%) were able to be serogrouped (Figure 3). This is a significantly higher proportion of cases than in 2001 $(83\%)^3$ or 2000^2 (73%) (p<0.003). Of the 117 isolates, 59 (50.4%) were serogroup B, 48 (41.0%) were serogroup C and 10 were other serogroups (5 were Y, 4 were W135 and 1 was X) (Table 3). Serogroup X has not been isolated from a sterile site (excluding conjunctivitis) in Queensland previously.

The percentage of isolates that were serogroup C is the highest since 1994; it is not significantly higher than 2001 or 1995–1996 (p=0.1) but is significantly higher than 1997–2000 inclusive (p 0.02) (Table 3).

There were three cases (11.5% of isolates) caused by serogroup C in children aged less than five years, although there were no cases caused by serogroup C in children aged under 12 months of age. In cases aged 15–19 years of age, 14 (51.9%) of the isolates able to be serogrouped were serogroup C (Figure 3). The proportion of disease due to serogroup C in these key age groups has not altered significantly in the last three years (p 0.2). In 2002, it amounted to a rate of serogroup C meningococcal disease in children aged 1–4 years, of 1.5 cases per 100,000 population compared with 5.3 cases per 100,000 population in the 15–19 age group.

Figure 3. Number of cases of invasive meningococcal disease, Queensland, 2002, by serogroup

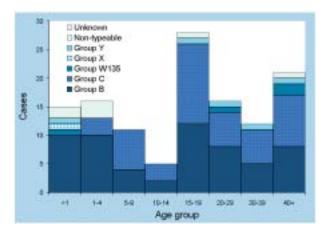


Table 3. Trends in invasive meningococcal disease serogroups, Queensland, 1994 to 2002

Year					Serogroup					Total
	E	3		С	А	W135	X	Y	Z	
	n	%	n	%	n	n	n	n	n	n
1994	41	55.4	32	43.2				1		74
1995	38	58.4	23	35.4	1	1		2		65
1996	45	59.2	23	30.3		4		3	1	76
1997	47	73.4	13	20.3		4				64
1998	57	71.3	11	13.8		6		6		80
1999	46	68.7	16	23.9		3		2		67
2000	37	77.1	10	20.8		1				48
2001	68	64.2	32	30.2		1		5		106
2002	59	50.4	48	41.0		4	1	5		117

When data were aggregated for the last four years, 1999–2002, 106 of 338 cases (31.4%) were serogroup C. During that time, there have been only two cases of invasive meningococcal disease due to serogroup C in infants under the age of one year. The proportion of disease due to serogroup C was significantly lower in the <1 year (5.1%) and the 1–4 years (15.8%) age brackets than any other age group (range: 32.0 % to 57.1%) (p<0.05) except for the 30–39 year age range (31.4%) where small numbers may affect the ability to detect significant differences. Almost half of the serogroup C cases (49 of 106 cases) occurred in persons aged between 15 and 29 years.

In 2002, serotyping and subtyping was performed on 86 isolates. The most common phenotype in 2002 was C:2a:P1.5 which comprised 23.3 per cent of all typed cases but more than a half of serogroup C cases (58.8%) compared with 25 per cent in 2001 (p=0.01). This relates to the ET15 strain which occurs among the C:2a:P1.5/P1.5,2 or ET37 lineage rather than to the actual phenotype C:2a:P1.5. Queensland first saw the ET15 strain in 1996 and it has been consistently present since but it was not possible to determine how many of the 2002 isolates were the ET15 strain.

The phenotype C:2a:P1.4, which is relatively new to Queensland, decreased from 16.7 per cent of isolates in 2001 to 11.8 per cent of isolates in 2002. The majority were identical to the Victorian strain (Helen Smith, personal communication).

The phenotypes B:4:P1.4 and B:NT:P1.4 accounted for 31 per cent of the typed serogroup B isolates; this is similar to 2001 (32%). There were three cases of phenotype W135:NT:P1.6. There were no links between these cases.

Outcome

There were four deaths from invasive meningococcal disease in 2002, representing a case fatality of 3.2 per cent. There was one death in each of the under one year, 5–9, 30–39 and over 40 years age brackets. There were two serogroup B (one B:NT:PT NT and

the other not able to be typed) and two serogroup C (C:2A:P1.4 and C:2A:PT NT) cases. The case fatality for cases with isolates of serogroup B was 3.4 per cent (2 of 59 cases) while it was 4.2 per cent (2 of 48 cases) for those with serogroup C isolates (p=1.0).

In 2002, the case fatality rate was the lowest in the four years of enhanced data collection. However, small numbers hamper the ability to discuss trends or analyse risk factors (Table 4).

Deaths during the four years of enhanced data collection were aggregated by age groups. The overall death rate in the period 1999 to 2002 was 7.5 per cent (31 of 411 cases). There was no significant difference in case fatality rates amongst the age groups (p=0.5).

Risk factors for dying of invasive meningococcal disease in 4 year period, 1999 to 2002

Because small numbers of fatal cases each year prevents the identification of significant risk factors for dying, information collected for the last four years has been pooled and analysed.

When data of cases in under 5-year-olds and over 30-year-olds were combined, this group was twice as likely to die from invasive meningococcal disease as those aged between 5 and 29 years in this 4-year period. Of all cases aged under 5 years or over 30 years, 10.2 per cent died compared with 4.9 per cent of 5–29 year olds but this difference was not significant (RR: 2.11; 95% CI: 1.02 - 4.37; p=0.06).

Outcomes according to gender and Indigenous status were not statistically significantly different (RR: 1.88; 95% CI: 0.91 - 3.89 and RR: 1.48; 95% CI: 0.48 - 5.54). Fatal outcomes were also not related to geographic location of the case (p 0.5) or to a history of overseas travel (RR: 1.17; 95% CI: 0.17 - 8.04).

Persons who presented with septicaemia alone were not at significant higher risk of dying compared with those who presented with meningitis alone (RR: 4.42; 95% CI: 0.54 - 35.90). However, those who presented with septicaemia with or without other clinical features were 11 times more likely to die than those who did

Table 4. Deaths due to invasive meningococcal disease, 1999 to 2002, by year

Year		Serogroup B		Serogroup C			All serogroups		
	Died	Total	%	Died	Total	%	Died	Total	%
1999	5	46	10.9	6	16	37.5	12	93	12.9
2000	1	37	2.7	1	10	10.0	4	66	6.1
2001	4	68	5.9	5	32	15.6	11	128	8.6
2002	2	59	3.4	2	48	4.2	4	124	3.2
Total	12	210	5.7	14	106	13.2	31	411	7.5

not have septicaemia as one of their presenting features (RR: 11.4; 95% CI: 1.6 - 82.6). Persons who presented with a petechial rash were 10 times more likely to die than those did not have a rash (RR: 10.1; 95% CI: 1.6 - 82.6). This may reflect stage of illness at time of reporting.

Over the four year period, cases due to serogroup C were 2.3 (95% CI: 1.11–4.82) times more likely to die compared with those caused by serogroup B.

General practice management and public health action

In 2002, information on timing of the management of invasive meningococcal disease was available for 80 cases. For these cases, median time delays at points of clinical progress are displayed in Figure 4 and Table 5. The median time taken from onset of illness to hospital admission was 19 hours (range: $\frac{1}{2}$ hour to 6 $\frac{1}{2}$ days) (Figure 4). The majority of cases were admitted to hospital within one day of the onset of their illness (Table 5); this was similar to 2000² (p=0.5) but faster than in 2001³ (p=0.002). In 2002, the median time for the four fatal cases to be admitted to hospital was not significantly different than for the cases who did not die (p=0.25).

Forty-nine (39.5%) of the 124 cases consulted a general practitioner (GP) about their illness; this included one of the fatal cases. Of these 49 cases, information was available on the suspected diagnosis for 38 of the cases. Almost a half (17 or 44.7%) identified the case may have had invasive meningococcal disease; 15 of these 17 (88%) were referred to the hospital by the GP at the time of consultation. For 21 cases, invasive meningococcal

Figure 4. Median time delays from meningococcal disease onset to notification and response

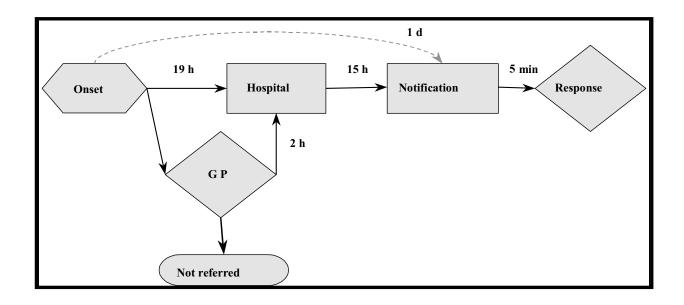


Table 5.	Median time delay	s from meningococcal	disease onset to notification and response

Intervals	Onset to hospital admission % (n=80)	Consultation to hospital admission % (n=29)	Hospitalisation to notification of PHU % (n=97)	Onset to notification of PHU % (n=84)	Notification to response by PHU % (n=108)
<1/2 hour	0.0	6.9	0.0	0.0	80.6
<1 hour	2.5	27.6	2.1	0.0	10.2
<6 hours	6.3	31.0	32.0	0.0	7.4
6–24 hours	62.5	17.2	33.0	32.1	1.9
24–48 hours	15.0	13.8	21.6	35.7	0.0
More than 2 days	13.8	3.4	11.3	32.2	0.0
Total	100	100	100	100	100

disease was not diagnosed at the time of the original consultation, but 18 of these 21 (86%) were referred to hospital; this included one fatal case. Of 41 who were referred by a GP to hospital, information was available on 29 cases. Of these 29, a majority were admitted within six hours of referral from the GP. This is similar to 2001^3 (p=0.7) (Table 5). In 2002, the median time between GP consultation and admission was two hours (range: <½ hour to 2½ days) (Figure 4). This is similar to 2001^3 (p=0.6).

However, in 2002 there was a significant difference in the time taken from GP consultation to hospital admission according to whether or not the GP diagnosed invasive meningococcal disease. The median time between consultation and admission was one hour (range: $<\frac{1}{2}$ hour to 21 hours) when this disease was considered and $10\frac{1}{2}$ hours (range: $1\frac{3}{4}$ hours to $2\frac{1}{2}$ days) when this diagnosis was not considered. This difference was significant (p=0.02).

Six persons (14.7% of those who consulted a GP) were given antibiotics prior to admission; this is a similar proportion to 2001^3 (21%) (p=0.2). Of these six, invasive meningococcal disease was considered in three cases, who were then promptly referred.

The majority of cases (67%) were notified to the relevant public health unit within a day of admission (Table 5). The median time between admission and notification was $15\frac{1}{4}$ hours (range immediately to $10\frac{1}{2}$ days) (Figure 4). This is similar to 2001^3 (p=0.5).

Sixty-eight per cent of cases were notified to the relevant public health unit within two days of onset of illness (Table 5). The median interval between onset and notification was 1 day $9\frac{1}{2}$ hours (range from $6\frac{1}{2}$ hours to 22 days) (Figure 4). This is not significantly shorter than 2001^3 (p=0.6).

Where information is available, the public health units initiated a response within six hours of notification for the majority (98.2%) of cases (Table 5 and Figure 4).

Discussion

Invasive meningococcal disease is a rare disease in Queensland, with an incidence of 3.4 cases per 100,000 population in 2002. This incidence was similar to 2001.

The completeness of the enhanced surveillance information provided by Public Health Units since its introduction in 1999 has improved, e.g. all cases had their Indigenous status identified in 2002. However, details about clinical presentation and clinical management can be further improved. There are concerns about ambiguity in some questions and inaccurate recording of data at the time of interview,⁸ and the enhanced surveillance form has undergone further modification as a result. Improved laboratory methods have an unmeasured influence on the measured incidence of disease. Improved laboratory methods also enable cluster identification.

The epidemiology of the disease was generally consistent with that seen in other years and trends are consistent with overall patterns of disease around Australia.⁹ There are some variations to note. The apparent decrease in incidence of invasive meningococcal disease amongst the 1–4 year age group and the concomitant increase in the incidence amongst the 15–19 year age group warrant continued surveillance to determine if this is a sustained change.

The percentage of isolates that were serogroup C has continued to rise and is the highest since 1994; it was statistically greater in 2002 than in the four year period, 1997–2000. The introduction of the meningococcal C vaccination program, particularly for the 15–19 year age group, is therefore timely.

The trend of decreasing case fatality rates over the last 4-year period is encouraging; further surveillance will determine if this downward trend is significant and sustained. Small numbers prevented the identification of any significant risk factors for dying in 2002. In the 4-year period during which enhanced surveillance has been conducted, the factors associated with an increased risk of fatal outcome were presentation with septicaemia, presentation with a rash and infection with serogroup C. Only one of the fatal cases consulted a GP prior to hospital admission, but did not receive antibiotics prior to admission. This suggests that cases with fulminant disease may present directly to hospital. We do not have information on other adverse outcomes to assess the effect of delays in obtaining medical attention.

It is well known that early presentation of invasive meningococcal disease can be variable and may not be severe. Indeed, only 66 per cent of cases notified in 2002 had a rash, emphasising (as in previous years^{2,3}) that absence of a rash cannot be considered to exclude the diagnosis of invasive meningococcal disease. The assessment of interval between onset of symptoms and hospital admission is difficult because the definition of onset of symptoms may differ due to this variability of presentation. As in previous years,^{1,2,3} septicaemia was the most common presentation; 76 per cent of septicaemic cases had a rash in 2002.

Less than half of the cases consulted a GP prior to hospital admission. Of those who did consult a GP, 41 (84%) were referred by the GP to hospital at the time of consultation. This is an increase from 2001³ when 51 per cent were referred by the GP to hospital at the time of consultation; although this may indicate improved clinical management, it may depend on the severity of the illness at the time of GP consultation. Severity of presenting illness is not collected in this enhanced surveillance. This enhanced surveillance indicated that a substantial number of patients were referred to hospital for further assessment even though the GP had not apparently made or was not convinced of the diagnosis of invasive meningococcal disease. To reduce ambiguity in seeking this information, the enhanced surveillance form has undergone further revision to clarify the seeking of this information. Diagnostic uncertainty, non-urgency of the case and the proximity of a hospital were explanations for not administering antibiotics prior to admission.⁸ Due to the small number of deaths, no conclusions can be drawn about the effects of the small number of cases given antibiotics prior to admission but theoretically, early antibiotic treatment is associated with decreased risk of adverse outcomes. The 2002 data does indicate that diagnosis of invasive meningococcal disease in a patient by the GP does expedite hospital admission. This issue will be reviewed in the analysis of enhanced surveillance data for 2003.

There is clearly a continued need to educate both the community and GPs about this disease to ensure that people seek early medical attention and are provided with early treatment to reduce the likelihood of adverse outcomes associated with this disease. Additional support may be needed to provide algorithms to assist GPs in reaching a greater confidence in diagnosis given the variability of the early clinical presentation of this disease.⁸ Although the small number of cases makes it difficult to determine if the reduction in case fatalities has a statistical correlation to more prompt medical attention, it is generally accepted that better outcomes occur when treatment is administered promptly.

In 2002, there was only one cluster of cases, illustrating that invasive meningococcal disease in Queensland remains a largely sporadic disease. A mass vaccination program was mounted as a response to this event incurring considerable costs. Public health services routinely follow up all cases of invasive meningococcal disease in order to ensure that all eligible contacts receive information on the disease and appropriate interventions. The response time after notification of a case continues to improve; there was a response mounted within six hours for over 98 per cent of cases in 2002.

Acknowledgments

All Public Health Medical Officers in Public Health Unit Networks as well as officers of the Communicable Diseases Unit contributed to this document. Data entry was performed by Cristina Chirico. Public Health Nurses in the Public Health Unit Networks also contributed through involvement with case investigation, public health responses and assistance with data collection. Queensland Health Scientific Services conducted the serogrouping, serotyping and serosubtyping. The staff of Queensland Health Pathology Services and the private laboratories are acknowledged for their contribution in the initial laboratory diagnoses across the state and the responsibility for almost all laboratory notifications in a very timely manner to the Public Health Units. General Practitioners and hospital staff together with the cases and their families provided the information.

References

- Ward J. Enhanced surveillance for meningococcal disease in Queensland in 1999. Queensland Health, 2000. Available from: http://www.health.qld.gov.au/ phs/Documents/cphun/6464.pdf
- Pugh RE. Meningococcal disease in Queensland, 2000. Queensland Health, 2002, Available from: http://www.health.qld.gov.au/phs/Documents/cdu/1 2776.pdf
- Pugh RE. Invasive meningococcal disease in Queensland, 2001. Queensland Health, 2002. Available from: http://www.health.qld.gov.au/phs/ Documents/cdu/13580.pdf
- Pugh RE. Invasive meningococcal disease in Queensland, 2002. Queensland Health, 2003. Available from: http://qheps.health.gov.au/phs/ Documents/cdu/19219.pdf
- Communicable Diseases Network Australia. *Guidelines for the early clinical and public health management of meningococcal disease in Australia*. Commonwealth Department of Health and Aged Care, 2001.
- Dean AG, Dean JA, Coulombier D, Brendel KA, Smith DC, Burton AH, et al. Epi Info, version 6.04b: a word processing, database, and statistics program for epidemiology on microcomputers. Atlanta, Georgia: Centers for Disease Control and Prevention, 1997.
- 7. StataCorp. Stata Statistical Software: Release 6.0. reference manual extract. College Station, TX: Stata Corporation, 1999.
- 8. Kari Jarvinen. Influencing general practitioner knowledge, attitudes and practice—first dose antibiotic administration for meningococcal patients. Queensland Health, 2003.
- 9. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2002. *Commun Dis Intell* 2003;27:196–208.

Appendix

Highlighted fields indicated by	Highligh	hted questions must be answered
Queensland Government Gueensland Health	MENINGOCOC PUBLIC HEALTH UNIT	CAL DISEASE CASE REPORT
		Hospital
Notified by		
Date initial response//	_ Time initial response_	am/pm
PATIENT DETAILS		
Patient's name		Phone:
		-
DOB// Ag	eyrsmos.	Sex [Male] [Female
Indigenous Status: I' Aboriginal	r TSI r Ab & T	SI I' Neither Ab or TSI I' Unknown
Occupation	Place of work/sc	haol
Preschool/child care	2013/07/2010/07/20	Phone:
CLINICAL PRESENTATION (Meningilis) TYes Petechial or purpuric rash	이거에 가지는 것이 많은 것이 없는 것이 것이 같이 것이 같이 것이 없다.	TYes I'No I'Unknown
		ther invasive illness (specify)
LABORATORY CRITERIA	(All highlighted details n	
solation of N meningitidis from CSF		ΓYes Γ No Γ Not done Γ Awaiting results
solation of N. meningitidis from blood	Construction of the Constr	Γ Yes Γ No Γ Not done Γ Awaiting results
solation of N. meningitidis from nasor		ΓYes Γ No Γ Not done Γ Awaiting results
solation of N. meningitidis from other specify site)	site	$\Gamma \mathrm{Yes} \ \Gamma \operatorname{No} \Gamma \operatorname{Not} \operatorname{done} \Gamma \operatorname{Awaiting results}$
Gram neg. intracellular diplococci in C	SE/blood	ΓYes Γ No Γ Not done Γ Awaiting results
N. meningitidis IgM+ve 🏾 🛛 Yes		tis IgM and/or IgG titres
Detection of meningococcal antigen (I specify site)	ADM COMPANY OF THE OWNER OWNER OF THE OWNER OWNE	ΓYes Γ No Γ Not done Γ Awaiting results
Detection of N. meningitidis DNA by P	CR in blood	ΓYes Γ No Γ Not done Γ Awaiting results
Detection of N. meningitidis DNA by P	A REAL POINT OF THE R	ΓYes Γ No Γ Not done Γ Awaiting results
	All highlighted details mus	
		onfirmed
ADDITIONAL LABORATORY I		
Serogroup ΓΑ ΓΒ ΓC Γ. Serotype Subtype		fy)b details
Serotype Subtype CLINICAL COURSE AND OUT	And the party of the local division of the l	u unana
Date of onset//		es FNo FUnknown
Time of onset	Date Died	
Was case referred to hospital by a	- 101 (P. 10) (P. 10)	lo E Unknown
yes, did GP consider meningocod		lo T Unknown
Date// time seen by G		
	12,72 million and the second second second	hospital ED
Hospital	an an an and so she	
Were parenteral antibiotics given p hospital admission?	100 10	No l'Unknown
IV/IM antibiotics	Date	// Time:

Appendix (continued)

the second se	NAGEMENT						
Were blood	cultures taken	before first d	ose antibiotics?	Γ Yes	ΓΝο Ι	Unknown	
Was throa	t swab taken a	t the time of fi	irst dose?	∏' Yes	ΓΝο Ι	Unknown	
(Antibiotics	used in hospital						
(Chemoprop	ohylaxis given to	patient?	Γ Not required	Γ Yes	ΓΝο Γ	Unknown	
RISK FAC	TORS		_				
Contact wit	h presumptive	meningococo	cal case in 60 days	before or	set I Ye	es 🛛 No	Γ Unknown
If yes, was	s prophylaxis of	fered?			ΓY	⊧s ∏No	Γ Unknown
If yes, was	s prophylaxis tal	ken?			Гү	es I No	[] Unknown
If yes, spe	cify type of prop	phylaxis			ГА	ntibiotic	Γ Vaccine
and the second	presumptive cas						
	ontact with press	States a second second			_(see contac	t management o	categories belov
Attends o	hild care / pres	school / schoo	ol / university		Г үе	219-2511-655	
Baturned	or arrived from		ountry in past 60 d		ΓYe	E No.	Γ Unknown
	factor for menin	a she was a second			1 11	10 1 140	1 00000000
OUTBREA	K DETAILS						
Constant of the local diversion	Contra and Contra and South	And in case of the local division of the loc	cases of the same	disease?	ΓYe	as ΓNo	
Is this case (known to be lin	And in case of the local division of the loc	cases of the same	disease?	ΓYe	as ∏No	
Is this case I	known to be lin	And in case of the local division of the loc	Number offered vaccine	disease?		es [] No	
Is this case i Details CONTACT Type of contact	known to be lin S Number of contacts	Number	Number offered	disease?			
Is this case i Details CONTACT Type of contact Household Child-care or	known to be lin S Number of contacts	Number	Number offered	disease?			
Is this case in the case in the case in the case is th	known to be lin S Number of contacts	Number	Number offered	disease?			
Is this case i Details CONTACT	known to be lin S Number of contacts	Number	Number offered	disease?			
CONTACT Type of contact Household Child-care or Preschool Close institutional Exposed to oral	known to be lin S Number of contacts	Number	Number offered	disease?			

Please Fax To CDU: (07) 3234 0057