In Canada, the western blacklegged tick, *Ixodes pacificus* and the blacklegged tick, *Ixodes scapularis*, are the principal vectors of *Borrelia burgdorferi*, the causative agent of Lyme borreliosis. These two tick species are also capable of transmitting *Babesia microti*, the pathogen which causes human babesiosis, although these tick-transmitted diseases can occur. However, the probability of these tick-transmitted diseases occurring is low. This is reflected in the low number of indigenous cases of Lyme borreliosis reported in Canada. Similarly, because *I. pacificus* and *I. scapularis* are not widely established in Canada, the probability that humans who are infected with *B. microti* elsewhere will serve as reservoirs of infection for local populations of these ticks is equally small. However, this does not negate the importance of clinicians in Canada being aware of the possibility that these tick-transmitted diseases can occur.

References

2. Gregson JD. The Ixodidae of Canada. Ottawa, Ont.: Canadian Department of Agriculture, Division of Entomology, 1956 (Publication 930);1-92.

Source: R Lindsay, PhD, H Artsoh, PhD, Zoonotic Diseases Section, Bureau of Microbiology, Laboratory Centre for Disease Control, Winnipeg, MB; I Barker, PhD, DVM, Department of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, ON.

DISTRIBUTION OF *IXODES PACIFICUS* AND *IXODES SCAPULARIS* RE CONCURRENT BABESIOSIS AND LYME DISEASE

In Canada, the western blacklegged tick, *Ixodes pacificus* and the blacklegged tick, *Ixodes scapularis*, are the principal vectors of *Borrelia burgdorferi*, the causative agent of Lyme borreliosis. These two tick species are also capable of transmitting *Babesia microti*, the pathogen which causes human babesiosis. In the present time, *I. pacificus* is endemic to localized areas of southern British Columbia in the vicinity of the Fraser Delta, and on the Gulf Islands and Vancouver Island. Similarly, reproducing populations of *I. scapularis* are currently only known to be established on the Long Point peninsula and Point Pelee National Park, both of which are located on Lake Erie, Ontario. Although specimens of *I. pacificus* have been collected at widely scattered localities in all provinces of Canada between Manitoba and Newfoundland, at most of these localities only individual or small numbers of adult ticks (usually females) have been encountered, usually on dogs or people. This suggests that these ticks have been adventitiously introduced to these widely scattered areas, likely on birds, but does not indicate that tick populations are established. Similar introductions of *I. pacificus* to non-endemic regions of British Columbia likely occur but have not been documented to date. Thus at the present time, established populations of *I. pacificus* and *I. scapularis* are focal and limited in Canada.

Although people or pets living outside of the tick-endemic areas of British Columbia and Ontario can be infested locally by an adventitious tick, and hence potentially infected with any of the pathogens associated with these species, the probability of this occurring is low. This is reflected in the low number of indigenous cases of Lyme borreliosis reported in Canada. Similarly, because *I. pacificus* and *I. scapularis* are not widely established in Canada, the probability that humans who are infected with *B. microti* elsewhere will serve as reservoirs of infection for local populations of these ticks is equally small. However, this does not negate the importance of clinicians in Canada being aware of the possibility that these tick-transmitted diseases can occur.
MANAGEMENT OF A CLUSTER OF CASES OF INVASIVE GROUP C NEISSERIA MENINGITIDIS INFECTIONS IN THE HAMILTON-WENTWORTH REGION – ONTARIO

Introduction

The Canadian Laboratory Centre for Disease Control (LCDC) held a Canadian Consensus Conference in February 1993 on meningococcal disease. Subsequently, the Advisory Committee on Epidemiology (ACE) revised its guidelines for the management of clustered cases of meningococcal disease to reflect the recommendations of the consensus conference[1].

Invasive meningococcal disease continues to be a source of public concern in communities across Canada. Of the 13 serogroups, groups A, B, C, Y, and W-135 are the most common. The incidence of disease is cyclical in nature, increasing in late winter and early spring in Canada. In 1985, 46% of isolates grouped were serogroup B and 24% were serogroup C. In 1992, 24% were serogroup B and 56% were serogroup C. In addition, beginning in 1989, the incidence of serogroup C in the age group 5 to 19 years old has more than doubled[1]. Outbreaks of serogroup C meningococcal disease have been occurring more frequently since the early 1990s and the use of meningococcal vaccine to control these outbreaks has also increased[2].

ACE recommends the administration of chemoprophylactic antimicrobials to all close contacts of a sporadic case of meningococcal disease but not to school contacts, transportation and workplace contacts, nor social contacts who are not close. In addition, where evidence suggests that an outbreak is occurring or where there is a cluster of cases in a delineated population caused by a vaccine-preventable serogroup, vaccination of all persons at risk should be considered. When two cases of the same vaccine-preventable serogroup occur in the same school within one month, one should immunize vaccine-eligible students attending the entire school[3].

This paper describes the application of the 1994 guidelines for the control of meningococcal disease in a school community in the Hamilton-Wentworth region in 1996, and presents a framework to assist in the management of a cluster of invasive group C infection in a school.

The terms below, as defined by the revised 1994 ACE guidelines, were used as a basis for public-health action[3].

A sporadic case of invasive meningococcal disease is a single case that occurs in a community where there is no evidence of an epidemiologic link (by person, place, or time) to another case.

A secondary case has had contact with a case and illness begins at least 24 hours after that in the index case. Cases developing within 24 hours of onset of illness in the index case are defined as co-primary cases.

Close contacts are household contacts of the case, contacts who share sleeping arrangements, child-care and/or nursery school contacts, and all persons with direct contamination of their nose or mouth with oral and/or nasal secretions of a case.

A cluster is two or more cases of the same serogroup that are closer in time and space than expected for the population or group under observation.

An outbreak is an increased transmission of Neisseria meningitidis in a population, manifested by an increase in cases, where there is an absence of links between cases, based on common personal contacts or a common place of exposure. The closer the association between cases, e.g. two cases who shared oral secretions, the less likely that the cases reflect an increase in the risk of transmission within the broader community.

Background

In the Hamilton-Wentworth region, the number of cases has remained stable at four to six cases per year, an incidence rate of 1.25 cases per 100,000, which is slightly higher than the provincial rate of 1 case per 100,000. During the period between January 1991 and December 1996 inclusively, 32 cases of meningococcal disease were confirmed (Table 1). Nine of the 32 cases occurred in individuals 13 to 19 years of age. Eight of the nine were identified as serogroup C; the other case had no growth on culture. The second largest number of cases occurred in children < 1 year of age, i.e. six of the 32 cases. Five were identified as serogroup B and the other as serogroup C.

<table>
<thead>
<tr>
<th>Year</th>
<th>Total number of cases</th>
<th>Rate per 100,000</th>
<th>No. of Serogroup C cases</th>
<th>No. and % of cases 13 to 19 years of age</th>
</tr>
</thead>
<tbody>
<tr>
<td>1991</td>
<td>5</td>
<td>1.25</td>
<td>2</td>
<td>1 (20%)</td>
</tr>
<tr>
<td>1992</td>
<td>4</td>
<td>1</td>
<td>3</td>
<td>2 (50%)</td>
</tr>
<tr>
<td>1993</td>
<td>6</td>
<td>1.5</td>
<td>1</td>
<td>1 (16.6%)</td>
</tr>
<tr>
<td>1994</td>
<td>5</td>
<td>1.25</td>
<td>2</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>1995</td>
<td>6</td>
<td>1.5</td>
<td>2</td>
<td>2 (33.3%)</td>
</tr>
<tr>
<td>1996</td>
<td>6</td>
<td>1.5</td>
<td>1</td>
<td>3 (50%)</td>
</tr>
</tbody>
</table>

* Hamilton-Wentworth Regional Public Health Department Reportable Disease Information System

Over a 6-week period between 26 September 1996 and 12 November 1996, three cases of invasive N. meningitidis disease were reported to the Regional Public Health Department (RPHD). All three were secondary school students attending two different schools and no apparent social contact had taken place between the three students.

Case 1

The first case was a secondary school student in the East Mountain area of the region. Onset of illness began 26 September 1996 when the case presented with high fever, vomiting, and lethargy. On 27 September, a rash appeared on the hands and feet. The case was admitted to hospital with gastroenteritis and sepsis on 28 September. Blood cultures taken after admission were
initially negative. However, these subsequently grew gram-negative diplococci identified as *N. meningitidis*, serogroup C. A specimen of cerebrospinal fluid (CSF) taken 29 September was clear and negative on gram stain and culture. The RPHD received the report on 30 September, and follow-up investigations took place immediately.

This case was considered a sporadic case at this time and prophylaxis was given, as per revised ACE guidelines, to all household members, which included two parents and a sibling, as well as to three close friends. This student fully recovered after intravenous antibiotic treatment.

**Case 2**

The second case was a secondary school student in the West Mountain area of the region. The school attended by this case was located approximately seven kilometres west of the school attended by Case 1. On 9 November 1996, this student developed high fever, lethargy, stiff neck, and headache, and was taken to hospital on 10 November. A specimen of CSF showed occasional gram-negative diplococci which were identified as *N. meningitidis*. The RPHD was notified that same day.

The case investigation produced many contacts and indicated involvement in many activities, including a student co-op placement in a day-care facility attached to the school. This investigation required many hours of assessment for extent of exposure and the need for prophylaxis. As per the revised ACE guidelines, nine individuals including household members and friends were identified as close contacts and received rifampin prophylaxis.

A preliminary report on 14 November identified the serogroup as B. However, 5 days later the laboratory reported that this result was premature. Confirmation of serogroup C was received 21 November 1996.

This case responded well to intravenous antibiotics and was discharged from hospital 9 days after admission. Of note is the fact that a sibling had been ill with the same infection 12 months earlier.

**Case 3**

The third case was a student attending the same secondary school as Case 2. The onset of illness was 12 November 1996, 3 days after that of Case 2. The case presented with symptoms including fever, vomiting, and a generalized rash, and was admitted to hospital on 13 November. A CSF specimen grew *N. meningitidis*. This case was reported to the RPHD on 13 November.

Case investigation of contacts required extensive assessment to determine the extent of exposure and the need for prophylaxis. A total of six household and close contacts received rifampin prophylaxis.

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**Public-health action**

For all three cases, public-health action was based on the 1994 revised ACE guidelines for the control of meningococcal disease. Case 1 was defined as sporadic and managed accordingly.

Prior to 21 November, before serogroup results were known, no evidence of contact was found between Cases 2 and 3. Public-health action included providing a letter and information fact sheet to all students at the school attended by Cases 2 and 3. Prophylactic antibiotics were recommended for individuals identified as close contacts. Active surveillance was initiated in the school as well as in area hospital emergency departments to identify any new cases. Public-health nurses called the attendance monitor at the school each morning for a report of all the students calling in sick and reviewed the presenting symptoms. Calls to the infection control officers in the hospitals helped to identify any potential cases seen in the emergency departments. Active surveillance continued for 10 days from the onset of symptoms for Case 3. No new cases were identified.

The day-care centre attached to the secondary school was advised not to place students from the school co-op program until further notice from the health department. The family studies department at the school was also advised not to place any students in any other day-care settings until after 22 November – the end of the incubation period (10 days post onset of symptoms) for any newly infected contacts of Case 3.

On 21 November, the RPHD received the final laboratory results for Cases 2 and 3 and confirmed them both as serogroup C. It became evident that this was a school-based cluster and this information immediately changed the planned public-health action.

That same day, an emergency outbreak control team was mobilized to plan and implement a meningococcal vaccine program for the school attended by Cases 2 and 3. The rationale was as follows: “When two cases of the same vaccine-preventable serogroup occur in the same school within one month, one should immunize vaccine-eligible students of the entire school. There is no evidence to support vaccination of adults at the school or home”[41].

The precautionary program was organized in consultation with the Disease Control Service of the Public Health Branch, Ontario Ministry of Health, and with the collaboration of the staff and managers of the RPHD. After consultation with the Disease Control Service, the medical and associate medical officers of health decided that the public-health department would offer meningococcal vaccine to the students at the school on 22 November; children at the day-care would also be included. As yet, there is no evidence available to determine whether attendance at a day-care centre increases the risk of acquiring meningococcal disease in young children[3]. However, the incidence of disease is high in this group and, due to the close association with the affected school, they were considered at risk for infection. Therefore, this population was included.

A team of 37 health department staff members was mobilized on the afternoon of 21 November for an urgent immunization
clinic the next day. All necessary preparation, including alerting the media, area physicians, hospitals, and other community partners, was completed late on 21 November. By 14:00 hours on 22 November, 1,357/1,569 secondary school students (86.5%) and 39/42 (93%) of the full-time day care children were immunized.

Four nursing staff attended the school on 25 November to offer vaccine to students who had been absent on 22 November, and to arrange for immunization opportunities of the part-time day-care children. An additional 56 secondary students and one more day-care child were immunized. In total, 1,413 (90%) secondary school students and 40 (95%) day-care attendees were immunized.

Discussion

Follow-up investigation to obtain important epidemiologic information along with timely and accurate results of all laboratory testing cannot be undervalued in a potential outbreak situation.

Initially, Case 1 was not considered part of the school-based cluster because of the 4-week time interval between Cases 1 and 2. However, all three cases were serogroup C, lived in the same geographic area, and were in the same age group. This information strengthened the decision to coordinate a school-based immunization program.

The final laboratory results, received 16 November 1996, showed that all three cases had similar phenotype with the same electrophoretic type (ET) and grouped into similar genetically related ET complexes. Therefore, even though the investigations found no apparent connection between the three students, the final results showed that a similar strain of meningococcal disease was circulating in the community.

The cost of the immunization program was approximately $43,740.00, based on average hourly rates for the staff and excluding vaccine costs and supplies.

Conclusion

In conclusion, planning and implementing this urgent program was made possible by the essential and important community partnerships already in place with the local hospitals, physicians, and the school. In addition, the collaborative consultation with the Public Health Branch in the interpretation and application of the revised ACE guidelines was an integral part in the management and prevention of an outbreak of meningococcal disease.

References


Source: O Tolomeo, RN, BSc, C Buffett, RN, BScN, MSc, E Richardson, MD, MHS, Communicable Disease Program, Hamilton-Wentworth Regional Public Health Department, Hamilton, ON. (Adapted from Public Health & Epidemiology Report Ontario 1998;9:90-95.)

International Notes

IMPORTED DRACUNCULIASIS – UNITED STATES, 1995 AND 1997

Dracunculiasis is a parasitic infection caused by a filarial worm (Dracunculus medinensis, i.e. Guinea worm) that is transmitted through contaminated drinking water. Approximately 1 year after a person is infected, one or more meter-long adult female worms begin to emerge through the skin, often incapacitating the patient for 2 months. Despite a dramatic decrease in cases worldwide, dracunculiasis is still occasionally imported into the United States. Since 1995, two cases of dracunculiasis have been reported in the United States, both imported from Sudan. This report summarizes the investigation of these cases.

Case 1

A 9-year-old girl residing in Tennessee had emigrated from Sudan in September 1995. Before the girl left Sudan, a Guinea worm had emerged and had been extracted from her right lower leg. The lesion had healed when she arrived in the United States. After she had been in the United States for 3 weeks, another Guinea worm began to emerge from her left leg. Medical examination at a local health clinic revealed a string-like worm dangling from a lesion on her left leg, and she was referred to an infectious disease specialist. The leg was secondarily infected and swollen, and the girl was unable to walk. Despite antibiotic treatment, her cellulitis did not improve, and the lesion was surgically opened, drained, and debrided of pus, necrotic debris, and fragments of the Guinea worm. The patient was hospitalized for 2 weeks, requiring surgery to stretch a contracture of her ankle and to apply a skin graft to the wound. After outpatient physical therapy, she was able to walk without crutches.

Case 2

A 31-year-old woman residing in Connecticut had emigrated from Sudan in January 1997. In April 1997, she was evaluated at a university clinic for possible tuberculosis (TB). A radiograph revealed lung lesions consistent with TB and a worm-like calcification in her left chest. Physical examination revealed multiple, indurated, oval lesions 4 cm to 8 cm in diameter on both lower legs. The patient reported the lesions had been present for 1 year and were intermittently painful. She recalled that a long string-like worm had emerged from her leg during the previous year. Biopsy of the leg lesions revealed erythema induratum, consistent with Bazin disease, a cutaneous manifestation of TB. The patient had evidence of a dead and calcified Guinea worm in her chest and a history suggesting a live Guinea worm had emerged from her leg before she arrived in the United States. She
also had pulmonary TB with a cutaneous tuberculid skin manifestation. Treatment with isoniazid, rifampin, and pyrazinamide resulted in elimination of acid-fast bacilli from sputum and resolution of cutaneous manifestations.

**MMWR Editorial Note**

No case of dracunculiasis transmitted in the United States has ever been reported, and importations of dracunculiasis to the United States are infrequent. Although both cases in this report involved refugees from Sudan, they differ in clinical manifestations and epidemiologic significance.

The risk for transmission of dracunculiasis from active cases imported to the United States is low; transmission would require a person with an emerging worm to enter a stagnant, freshwater pond containing copepods, and persons to drink directly from the source within 1 week after contamination. The disease can be completely prevented by keeping infected persons from entering and contaminating the water supply or by providing drinking water free of *Dracunculus medinensis* larvae. Humans are the only vertebrate host for *D. medinensis*. Only the worm that emerged from Case 1 could have posed any risk for contaminating a source of water in the United States. The calcified worm and the history of an emerging worm in Case 2 reflected previous infections without any possibility of transmission in the United States.

Although no drug aborts dracunculiasis infection or hastens expulsion of the adult worm, compounds that reduce inflammation and antibiotics to treat secondary infection facilitate extraction. Dracunculiasis treatment has included cleaning of the lesion and gentle traction to draw the long worm through the skin; the process may take several weeks. Care must be taken to avoid breaking the worm under the skin and subsequent allergic reaction to the internal components of the worm. Physicians who treat patients who have imported dracunculiasis can obtain treatment advice from the United States Centers for Disease Control and Prevention (CDC).

The global campaign to eradicate dracunculiasis began in 1986; the number of cases worldwide decreased by > 95% (from approximately 3.2 million cases in 1986 to 152,805 in 1996). Ongoing transmission of dracunculiasis is limited to 16 countries in Africa. In Asia, the disease is still occurring in Yemen. In India, the only other Asian country not yet declared free of dracunculiasis, no cases have been reported since July 1996. Pakistan, which reported its last case in October 1993, was certified free of dracunculiasis by the World Health Organization in 1997. In comparison with the dramatic decrease in cases and in villages with endemic disease globally, the numbers of reported cases and villages with endemic disease in Sudan increased sharply from 1993 to 1996. The areas with the highest prevalence of dracunculiasis are in southern Sudan, where war hampered surveillance for cases and interventions. In 1996, the 64,608 cases reported from areas in Sudan where surveillance was possible accounted for 78% of all cases worldwide.

The detection and investigation of every active case brought to the United States enables identification of places where dracunculiasis may still be present and prevents establishment of a focus of transmission in the United States. CDC requests that medical practitioners report any cases of dracunculiasis in the United States since 1990. A brief description of the case, including where the patient may have acquired dracunculiasis, location of treatment, approximate date of worm emergence, and clinical outcome should be reported to Guinea Worm Cases, Division of Parasitic Diseases, National Center for Infectious Diseases, CDC, Atlanta, GA 30333; telephone (770) 488-4531; or by e-mail at kdkl@cdc.gov.

**References**
