Meningitis in the central Arctic: a 4-year experience

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There were 37 cases of meningitis during a 4-year period among the native and white populations served by the Churchill Health Centre in northern Manitoba, an annual incidence of 128 per 100 000 in the overall population and of 202 per 100 000 among the Inuit. Bacterial meningitis predominated; *Neisseria meningitidis* and *Haemophilus influenzae* each accounted for one third of the cases. There were five deaths, and 14 of the survivors had severe sequelae. Therefore, although the doctors and nurses involved in the study had improved access to telecommunication and air transportation services in caring for patients in isolated northern settlements, and despite their efforts to be vigilant for possible cases of meningitis and to begin vigorous treatment early, the incidence, morbidity and mortality of this disease remained relatively high, particularly among the Inuit.

Au cours d'une période de 4 ans on a enregistré 37 cas de méningite parmi des populations indigène et blanche desservies par le Churchill Health Centre dans le nord du Manitoba. Ceci représente une incidence annuelle de 128 par 100 000 pour l'ensemble de la population et de 202 par 100 000 parmi les Inuits. Les méningites bactériennes prévalaient, avec *Neisseria meningitidis* et *Haemophilus influenzae* comptant chacun pour un tiers des cas. On a compté cinq décès, et 15 des survivants ont conservé des séquelles modérées ou graves.

Les médecins et les infirmières participant à cette étude bénéficiaient d'un meilleur accès aux services de télécommunications et de transport aérien desservant les communautés isolées du nord. Néanmoins, et malgré les efforts pour dépister les cas de méningite possibles et mettre immédiatement en route un traitement vigoureux, l'incidence, la morbidité et la mortalité de cette maladie est demeurée relativement élevée, particulièrement chez les Inuits.

Meningitis is thought to occur in the population of Canada's northern areas more commonly than in southern communities. As well, because the scattered population makes precise diagnosis and early treatment of this condition difficult, it seems to cause more frequent and serious sequelae. A prospective study, covering the 51 months of Dr. Wotton's tenure with the northern medical unit of the University of Manitoba (October 1972 to February 1977), was undertaken to determine to what extent these observations applied to the catchment area of the Churchill Health Centre. During that time 37 cases of clinical meningitis occurred in the communities served by the centre.

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Study population

Besides serving 2500 local white residents and 600 Indians and Métis living in northern Manitoba, the Churchill Health Centre is also a referral centre for 3500 Inuit living in settlements in the Keewatin district of the Northwest Territories (Fig. 1). The Belcher Islands settlement was only in the health centre's catchment area for the first year of the study. Because the growth rate of the Keewatin population presently exceeds the national rate, the numbers we use are estimates taken at the midpoint in the study. The population of the settlements varied in number from less than 150 people in Whale Cove to 900 in Rankin Inlet, with a high proportion of young people; approximately 50% were younger than 16 years of age. The population of Churchill itself was relatively stable during the study. The Indian population included 200 Chipewyan living at Tadoule Lake, a community 240 km west of Churchill accessible only by plane, 100 Cree living in isolated small communities along the Bayline Railway

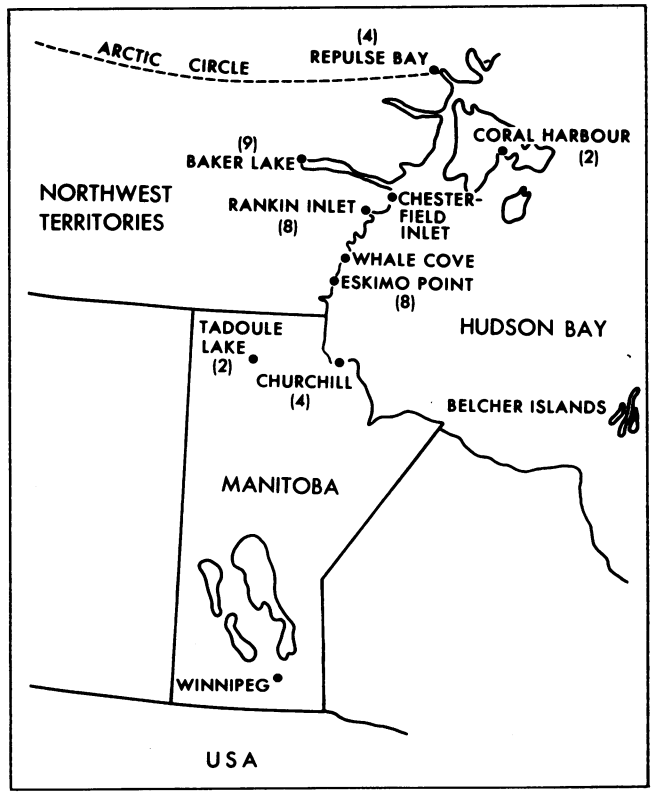

FIG. 1—Settlements in the Keewatin district of the Northwest Territories and in northern Manitoba served by the Churchill Health Centre. Numbers in brackets represent meningitis cases from each community.

between Gillam and Churchill, and 300 Cree–Métis living in Churchill.

The Inuit communities are provided with primary health care at nursing stations staffed by the medical services branch of the Department of National Health and Welfare. The stations have up to four nurses, depending on the size of the settlement, who function as nurse–practitioners and who maintain contact, by telephone, with physicians in Churchill. Seriously ill patients and those with complicated conditions requiring consultation, further investigation or hospitalization are evacuated from the Keewatin settlements to the hospital in Churchill, which is staffed by general practitioners. Tertiary care is provided by the teaching hospitals in Winnipeg. The distances some patients have to travel are immense: the flight from Repulse Bay, the most northerly settlement, to Churchill is 960 km, and it is another 960 km from Churchill to Winnipeg. Bad weather, such as a storm or a "white-out", or non-availability of aircraft can delay the evacuation of patients for days.

At the stations nurses are encouraged to be on the alert for possible cases of meningitis and to initiate antibiotic therapy early. When a delay in transporting a patient to hospital is encountered or anticipated, the station nurses may obtain cerebrospinal fluid by a lumbar puncture for diagnostic tests, and may begin to administer antibiotics intravenously. During our study more than 50% of patients arriving in Churchill had received some form of antibiotic therapy before they were assessed in hospital.

The Indians at Tadoule Lake have a health care worker who discusses patients with medical personnel by radio phone and organizes patient evacuations by air. At the time of our study people living along the Bayline Railway caught the train, which ran every other day, and presented at the outpatient clinic or emergency ward of the Churchill hospital.

Methods

Case definition

For this study we defined a condition as meningitis if the patient had clinical signs and symptoms that compelled specific treatment for meningitis, even if this diagnosis was not confirmed by tests of the cerebrospinal fluid. There were positive findings in the cerebrospinal fluid of 30 of the 37 patients: 11 had positive cultures, 9 had positive smears (8 of the 9 had other abnormalities in the fluid), 6 had high leukocyte counts and raised levels of protein, 1 had had a positive culture previously and was considered to be having a relapse, and 1 had gross cloudiness of the fluid (this patient was identified as having meningitis by a visiting physician at a nursing station where there was no laboratory that could confirm the diagnosis before treatment was started); in 2 patients the diagnosis was made from cerebrospinal fluid collected at autopsy. The remaining seven cases were diagnosed on other grounds: four patients had neurologic signs, one had petechiae and convulsions, one had a headache and a sore neck, and in one symptoms recurred (a blood culture at this time was positive).*

Table I—Incidence of bacterial meningitis in catchment area of Churchill Health Centre

Race	No. of patients during study	Population	Annual incidence (cases per 100 000)
Inuit	30	3500	202
Indian	4	600	157
White	2	2500	19
Overall	36*	6600	128.3

*One patient had a relapse during the study, giving a total of 37 cases.

Laboratory methods

Blood cultures and smears, and cultures, cell counts and biochemical analysis of the cerebrospinal fluid were done in the Churchill Health Centre clinical laboratory with standard techniques. The microbiology laboratory of the Health Sciences Centre in Winnipeg served as a reference laboratory and its director acted as a consultant.

Treatment

Our initial treatment regimen consisted of the intravenous administration of ampicillin, 400 mg/kg·24 h. Although no ampicillin-resistant *Haemophilus* organisms were cultured from our patients, because of the two patients with apparent relapse and in view of reports in the literature of the emergence of such organisms, chloramphenicol (150 mg/kg·24 h given intravenously) was added to the regimen in mid-1975 and administered until the culture reports were back, usually for 48 hours. When infections were positively identified as meningococcal or pneumococcal, we considered switching to intravenous penicillin G therapy. The intravenous antibiotic therapy was continued for 10 to 14 days.

Results

It is unlikely that any cases of meningitis were missed in our survey, as all records of death in the catchment area of the Churchill Health Centre were regularly reviewed. Of the 37 cases identified, 36 were bacterial and 1 was viral. Of the 36 patients with bacterial meningitis, 30 were Inuit, giving an annual incidence of 202 per 100 000 for this population (Table I). Indians and whites accounted for four and two cases respectively, giving annual incidence rates of 157 and 19 per 100 000 respectively. The average annual incidence for all the populations served by the Churchill Health Centre (128.3 per 100 000) contrasts sharply with the average for a typical county in the United States (7.3 per 100 000[1]) and is also higher than the incidence for Alaska (94.2 per 100 000) reported for 1971.[2]

The 37 cases were fairly evenly distributed throughout the 4 years of the study, and analysis of the cases

*A table showing details of the laboratory and clinical findings in all 37 cases is available from the authors.

month by month did not reveal any seasonal pattern. Nor was there geographic clustering within a community; the communities were evenly represented, the greatest number of patients coming from the two largest settlements.

Although the patients ranged in age from 2 months to 20 years, half of the cases occurred in the first year of life and only four patients were over 10 years of age. No neonatal infections were diagnosed.

Causative organism

Two causative organisms, *Neisseria meningitidis* and *H. influenzae*, each accounted for approximately one third of the cases of bacterial meningitis in our study. Of the remaining 12 cases, 11 were considered to be bacterial because of findings other than the results of cultures of cerebrospinal fluid; that is, purulent cerebrospinal fluid, organisms seen in smears of the fluid, low levels of glucose in the fluid, positive blood cultures or combinations of these findings.

H. influenzae was recovered in 9 of the 13 cases identified as being caused by this organism: the organism was cultured from the cerebrospinal fluid in 7 cases and from the blood in 2 cases. Of the remaining four cases, two were diagnosed on the basis of demonstration of the organism in a cerebrospinal fluid smear only. The other two cases may have been recurrences. In one child meningitis was diagnosed when *H. influenzae* was found in a culture of cerebrospinal fluid 1 month before our study began. He had received a full course of treatment with ampicillin given intravenously, but returned with symptoms of meningitis 8 weeks later. This time a culture of his cerebrospinal fluid gave negative results. A second child also received a full course of treatment with ampicillin given intravenously for bacterial meningitis although a culture and a smear of his cerebrospinal fluid had been negative. He was brought back with signs of meningitis 9 days after being discharged. Cultures and smears were again negative, but a diagnosis of meningitis was made because *H. influenzae* was isolated from a blood culture. With these two cases included, meningitis due to *H. influenzae* had an annual incidence in the total study population of 46.3 per 100 000, which exceeds the incidence in the United States of 4.0 per 100 000.[1]

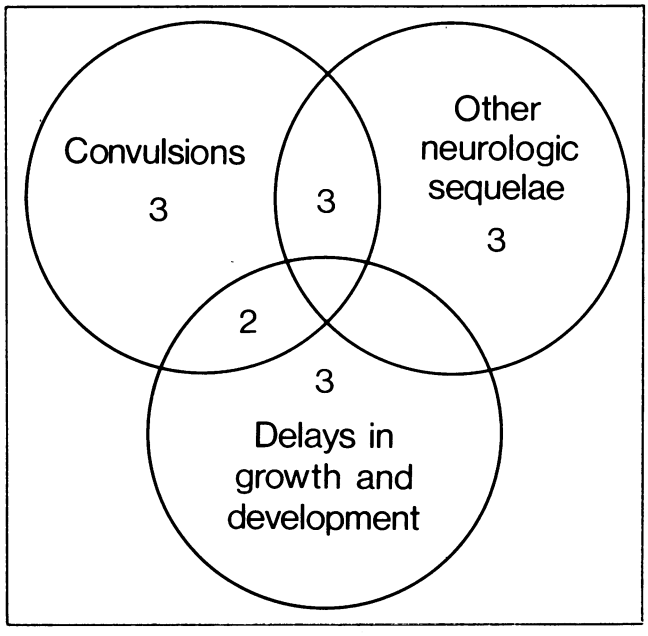

FIG. 2—Number of patients with various sequelae following their recovery from meningitis.

Nor were all of the 24 cases identified as having other specific bacterial causes so identified by culture of the cerebrospinal fluid. In three of these cases the patient's condition was identified as meningococcal meningitis after antibiotic treatment had been given: two of these patients had a positive smear of the cerebrospinal fluid and the third had a typical petechial rash.

Three cases were diagnosed at autopsy, including two cases of fulminating meningococcal infection and one case of *Bacteroides* infection in which the patient died from a temporal lobe abscess. Of the three cases diagnosed solely on clinical grounds, a typical petechial rash suggested a meningococcal infection in one case despite negative results from culture and Gram-staining of the cerebrospinal fluid, and symptoms and signs recurred within 2 to 8 weeks after treatment for *H. influenzae* meningitis in the other two cases.

Of the 37 patients we saw, 21 had had treatment with antibiotics before admission to hospital, usually one to four intramuscular doses. Eight patients had been given ampicillin intravenously for as many as 3 days before they were evacuated to Churchill.

Outcome

In five of the cases seen during our study the patients died. The status of the survivors was determined in mid-1978, a minimum of a year and as much as 5 years after their infection. We were unable to assess two of the patients. Almost half the survivors had severe residual disability. Eight suffered convulsions, six had other neurologic sequelae and five showed evidence of delays in growth and development; five had more than one of these sequelae. The interrelations are shown in Fig. 2.

Discussion

The findings in this study were different, especially for meningococcal infection, from those of other surveys of meningitis in native peoples. The annual rates of meningitis per 100 000 by causative organism among Navajo Indians,[3] Alaskan Inuit and Indians,[2] and our mixed study population were: *H. influenzae* 17.7, 62.8 and 46.3; *Diplococcus pneumoniae* 8.0, 19.3 and 7.1; and *N. meningitidis* 2.0, 2.4 and 42.8 respectively.

During the study period there was no recognizable meningococcal epidemic in either the Keewatin district or the Churchill area, and we have no explanation for the unusually high incidence of this disease. An earlier survey for meningococcal carriers in the population of Baker Lake in the Keewatin district showed a carrier rate of 9.6%.[4] The incidence rates of *H. influenzae* and pneumococcal meningitis in our population were considerably higher than those reported for one white and several black populations, in which the incidence

was 1.6 to 4.3 per 100 000 for the former organism and 0.9 to 4.9 per 100 000 for the latter.[1,5-7] The incidence of meningococcal infections in these populations was 0.8 to 13.5 per 100 000.

The mortality in this study was 14%, which is considerably higher than the mean rate of 8.7% for children 4 weeks to 10 years of age reported by Goldacre[8] for a large population in northwest London and adjacent counties, but closer to the 16.0% for Navajo Indians reported by Coulehan and associates.[3] Feldman and coworkers[9] have documented a higher mortality for black and Indian patients than for white patients with meningitis in the United States; this discrepancy seems to apply when Inuit and Indian patients are compared with white patients in northern Canada. On the other hand, death occurred in only 1 of 39 patients treated at the United States Public Health Service Alaska Native Hospital in Bethel in southwestern Alaska.[2] The higher mortality among native people is often blamed on their lack of access to optimal medical care. A recent review of race-specific rates of death from bacterial meningitis in the United States was unable to show that the higher rate in blacks and Indians was related to factors associated with race, such as poverty and crowding.[9] However, that survey was carried out 3 months after a community preventive treatment program using sulfonamide and within 2 days of a second course of treatment for children 6 years of age and under.

The fact that only one case of viral meningitis occurred in the 51-month period of our study contrasts with the common idea that viral meningeal disease is more prevalent than bacterial.

The incidence of bacterial meningitis was higher among the Indian and Inuit people of northern Manitoba and the Keewatin district of the Northwest Territories than would be expected from public health statistics for the United States; the incidence of meningococcal meningitis was particularly high. In spite of health care that provides improved telecommunications, rapid air transportation of patients, up-to-date diagnostic laboratory facilities and early vigorous treatment, meningitis remains all too common a cause of death among children in the North. Evidence that protection may be afforded, at least in children over 18 months of age, by pneumococcal,[10] *H. influenzae* type b,[11] and groups A and C meningococcal polysaccharide vaccines[12,13] supports the introduction of trial vaccine programs in these northern communities.

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Medicine teams with technology

The physician must look beyond the picture, now widely cherished, of the lonely, great-hearted doctor, bowed impotent beside the dying child. Modern transportation, modern methods of communication, and modern techniques of diagnosis and treatment, have made that picture as obsolete as the village blacksmith at his charcoal forge.

—William Dock) (1889–); "The Next Half Century in Medicine" (symposium)