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hpsc
Health Protection Surveillance Centre
25-27 Middle Gardiner St
Dublin 1, Ireland
Ph +353 1 876 5300
Fx +353 1 856 1299
E info@mailx.hse.ie
www.hpsc.ie

TB Outbreak Linked to Two Crèches

Summary

The most significant TB outbreak reported in Ireland in recent years is currently being investigated in Cork. The outbreak centres on two crèches. To date, 18 children and two adults (excluding the index case) have been identified. The vast majority of child cases are toddlers (children aged 2-3 years).

Introduction

A symptomatic crèche worker (index case) was diagnosed with sputum positive pulmonary TB in March 2007. The newly notified case had worked in two large crèches – in Crèche I for just over one month up until early March'07, and in Crèche II for over two years up until December 2006. The index case had worked primarily with toddler children in both crèches.

Screening

All exposed children and adult workers in both crèches have undergone screening (Round 1). In view of the extent of early findings, chest x-rays were included as part of the screening criteria for Round 1 in both crèche populations, regardless of Mantoux test results. Mantoux negative asymptomatic children have been offered isoniazid prophylaxis ('window prophylaxis') until the second round of screening has been completed and the overall results assessed.

Findings to date

TB case findings to date on the two crèche populations are summarised in table 1. Eighteen children and two adults (excluding the index case) have been identified. Twenty-nine per cent of the child population in Crèche I and 19% in Crèche II had evidence of BCG vaccination. None of the 18 child cases had BCG vaccination. All children, other than cases, with Mantoux ≥6mm and regardless of BCG status, have been offered treatment for latent TB infection (LTBI) and are under the care of a consultant paediatrician. Similarly, adult workers with Mantoux ≥6mm (other than cases) have been offered treatment for LTBI.

Table 1. Crèche populations: TB cases

	Crèche I Children 127/Staff 33	Crèche II Children 141/Staff 33	Total
Child cases	6	12	18
Staff cases*	0	2	2
Total	**6**	**14**	**20**

*Excluding index case

Extended screening

In addition to the crèche populations above, other cohorts were identified for screening including visiting cohorts of parents/minders/other children who would have been regularly collecting and dropping off toddler children at the toddler room. These cohorts were included as they may have had a significant cumulative close contact with the index case. Screening was also extended back to January 2006 in Crèche II given the degree of obvious infectivity of the index case. No further cases have been detected to date in any of those cohorts.

Investigation ongoing

The *Mycobacterium tuberculosis* strain isolated from the index case was reported to be pan-sensitive. The investigation of this unprecedented outbreak is being overseen by an outbreak control team and expert advisory group. The second round of screening will be commencing shortly.

In April 2007, the Primary Community and Continuing Care Directorate HSE South announced that all newborn babies in Cork will, from October 2007, be routinely offered BCG vaccination.

Margaret O' Sullivan and Elizabeth Keane on behalf of the outbreak control team and expert advisory group and all staff involved at the Department of Public Health, Cork.

Invasive Meningococcal Disease in Ireland, 2006

Introduction

Invasive meningococcal disease (IMD) was first described in 1805 during an outbreak in Switzerland. It was another eighty years before the causative agent, the organism we now know as *Neisseria meningitidis*, was identified. In the early 20th century up to 80% of people contracting IMD died from the disease. Mortality rates were reduced to approximately 25% with the introduction of anti-meningococcal serum therapy. This therapeutic approach later fell by the wayside due to its unpleasant side-effects and particularly with the advent of sulphonamide therapy. Antibiotics became the cornerstone in the management and treatment of IMD infections from the 1940s onwards, dramatically reducing mortality.

N. meningitidis is an encapsulated organism and infects only humans. It is classified into 13 serogroups on the basis of the chemical composition of the capsular polysaccharides. Five serogroups (A, B, C, Y and W135) are most commonly associated with invasive disease. Knowledge of the serogroup distribution of strains is useful information for vaccine development. The latter half of the 20th century saw a focus towards vaccine development and the use of vaccination as a means of IMD prevention and control. The development of monovalent, bivalent and tetravalent polysaccharide vaccines against serogroups A, C, W135 and Y and later conjugate vaccines against the same serogroups of *N. meningitidis* were important advances. The development of an effective vaccine against *N. meningitidis* serogroup B remains elusive.

Despite our improved understanding of the organism and advances in the prevention, diagnosis, and treatment of IMD, the disease is still a leading cause of meningitis and septicaemia in children and young adults and is a significant cause of mortality. An estimated 500,000 cases and 50,000 deaths occur per year worldwide.[1] In developing countries and particularly in sub-Saharan Africa, serogroup A is hyperendemic with incidence rates of up to 20 per 100,000 population occurring annually. Serogroup A and more recently serogroup W135 are frequently associated with epidemics in this region, stretching from Senegal to Ethiopia. During these epidemics, nationwide attack rates ranging from 100 to 800 per 100,000 population have been reported.[1] In contrast, in most developed countries endemic incidence rates of IMD range from <1 to 5 per 100,000 population with serogroups B and C being responsible for many of these cases.

The epidemiology of IMD has evolved in Ireland over the last 10 years. In the late 1990s, the disease was hyperendemic in this country. Annual incidence rates of almost 15 cases per 100,000 population were occurring which was the highest in Europe.[2] At that time serogroup B accounted for almost two-thirds of the cases and serogroup C one-third. The introduction of the meningococcal C conjugate (MenC) vaccine in October 2000 to the routine childhood immunisation schedule and a catch-up programme for under 23 year olds saw a 75% reduction in serogroup C disease within one year.[3] By the end of 2003, the incidence of serogroup C disease had declined by 96% compared with the pre-MenC vaccine era. As a result of this huge decline and coupled with a steady decrease in serogroup B cases over recent years, the overall incidence of IMD in Ireland has declined to approximately 5 cases per 100,000 population annually.[3] Ireland has emerged from the hyperendemic period and IMD is presently at endemic levels.

In this paper the epidemiology of IMD in Ireland in 2006 is reviewed.

Materials and Methods

IMD is a notifiable disease in Ireland, initially under the category 'bacterial meningitis (including meningococcal septicaemia)' and more recently as a specific disease in its own right when the Infectious Diseases (Amendment) (No.3) Regulations 2003 (SI No 707 of 2003) came into force on 1st January 2004.

Case-based data from the IMD enhanced surveillance system are available nationally since 1999 and all these data are on the Computerised Infectious Disease Reporting (CIDR) system. Details of IMD cases including information notified by clinicians and/or laboratories to medical officers are entered on to CIDR either by the regions where CIDR has been implemented (n=6) or by the Health Protection Surveillance Centre (HPSC) for the regions yet to implement the system (n=2). These data on CIDR are reconciled monthly by HPSC with the Irish Meningococcal and Meningitis Reference Laboratory (IMMRL) national database on laboratory-confirmed cases. Departments of public health are requested once per quarter to review IMD events on CIDR to ensure data are accurate and complete for that time period. At the end of each year, detailed cleaning and validation of data on CIDR is undertaken by HPSC in conjunction with the departments of public health and IMMRL.

For this paper data analysis was performed using CIDR and MS Excel. Incidence rates were calculated using the 2006 Census of Population as the denominator for the 2004-2006 data, the 2002 census for 2000-2003 data and the 1996 census for 1999 data.

Data for this paper were extracted from CIDR on 15th May 2006. These figures may differ from those published previously, due to ongoing updating of notification data on CIDR.

Results

Cases due to IMD

In 2006, 209 cases (4.9 per 100,000 total population) of IMD were notified in Ireland which was a very slight increase from 2005 when 203 cases (4.8 per 100,000 total population) were notified. One hundred and seventy three of the notifications in 2006 were classified as definite (82.8%), six as presumed (2.9%) and 30 as possible (14.3%) (table 1). In total, 87.5% (183/209) of the notifications were laboratory confirmed. Sixty one percent were diagnosed by PCR only, 25% by both PCR and culture, 9% by culture only, 3% by serology and 2% by microscopy. The usual seasonal decline of IMD seen during the summer months was observed in 2006, with just 12% of cases occurring in quarter three (Jul-Sept), while 35% of cases occurred in quarter one (Jan-Mar).

IMD by age

In 2006, more cases occurred in males (n=127) than in females (n=82), giving a male female ratio of 1.5:1.0. Cases ranged in age from one month to 84 years, with a median age of two years. Over half the cases occurred in children less than three years of age (figure 1). The age-specific incidence rate was highest in infants <1 year of

Table 1. Number of cases of IMD notified in Ireland by serogroup and case classification, in 2006

	Definite	Presumed	Possible	Total
Serogroup B	163	5	1	169
Serogroup C	4	0	0	4
Serogroup W135	1	0	0	1
Serogroup Y	4	0	0	4
Non-groupable (NG)	1	0	0	1
No organism detected	0	1	29	30
Total	173	6	30	209

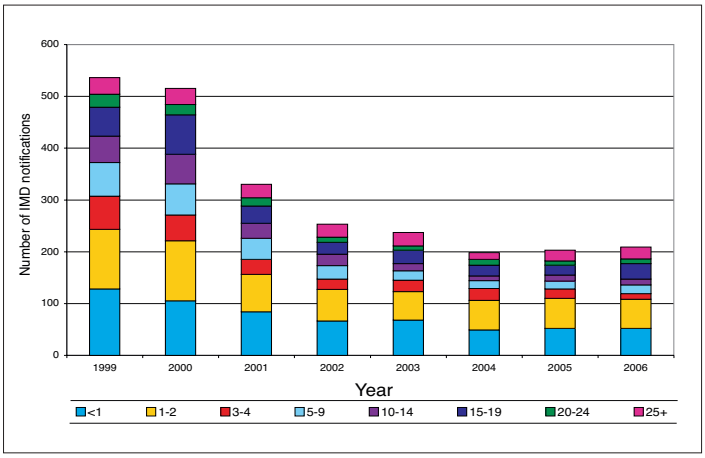

Figure 1. Number of IMD cases notified in Ireland by age group and year, 1999-2006

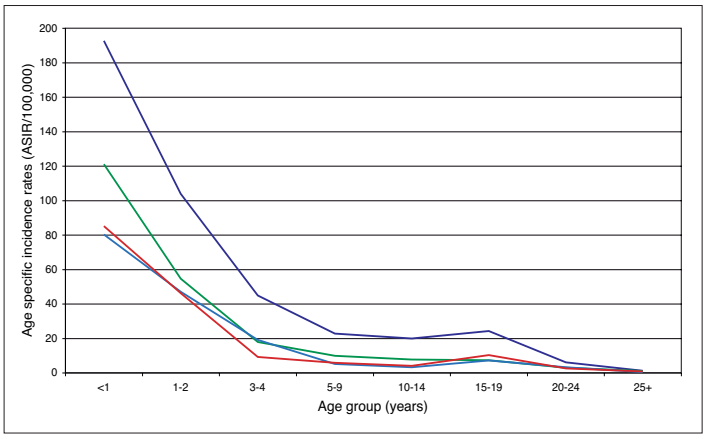

Figure 2. Age-specific incidence rate of IMD notifications in Ireland, in 2000, 2002, 2004 and 2006

age (85.1 per 100,000) followed by the 1-2 year olds (46.2 per 100,000) (figure 2). Thereafter, the incidence rates decline considerably in the older age groups (figure 1 and figure 2). Since 2004, the distribution of and incidence of cases by age group have been very similar. The exceptions in 2006 were (i) in the 3-4 year olds when a decline in incidence was observed compared to previous years and (ii) a slight increase in incidence in the 15-19 year old age group was noted (figure 1 and figure 2). The incidence of IMD in all age groups under 25 years of age had declined by over 55% in 2006 when compared with 2000 (figure 2).

IMD by serogroup

N. meningitidis serogroup B was the causative agent in the majority of the IMD cases notified in 2006, accounting for 81% (n=169) of the cases (table 1). The remainder of the cases were due to serogroup C (n=4), serogroup Y (n=4), serogroup W135 (n=1), and non-groupable (n=1). Thirty cases had no organism detected and 29 of these were classified as possible IMD cases (table 1). Of the four serogroup C cases one occurred in a young child, the remaining three in adults. The child involved had been fully vaccinated against serogroup C disease and therefore the case constitutes a MenC vaccine failure, the only one in 2006. The distribution of non-serogroup B/C cases has not changed greatly over the last eight years, with serogroup W135 and serogroup Y cases ranging between one and six cases per annum. Each year since 2003, serogroup B accounted for 80% and over of the IMD cases, serogroups C, W135 and Y when combined accounted for <10%, and cases where no organism was detected accounted for 10% and over (figure 3).

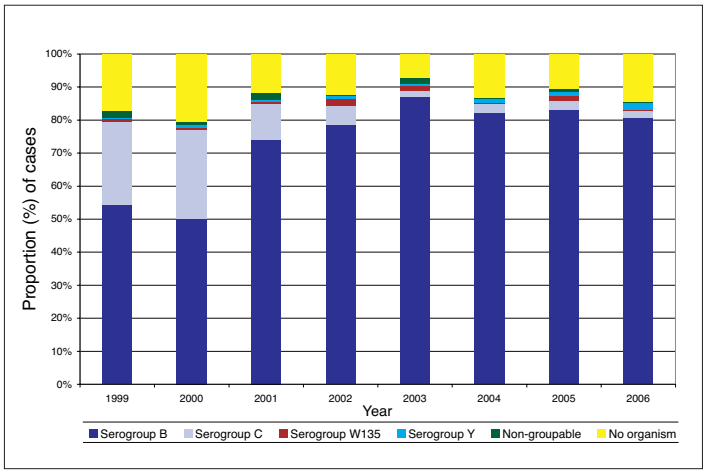

Figure 3. Percentage distribution of IMD cases by serogroup, 1999-2006

Deaths due to IMD

The case fatality ratio (CFR) due to IMD in 2006 was 2.4%, with five deaths reported. All deaths were due to serogroup B (CFR of 3%). Four occurred in children under five years of age and one in a young adult under 25 years of age. Therefore, the CFR due to serogroup B IMD was 4.2% in children <5 years of age. This is the second consecutive year where no serogroup C deaths occurred. In 2005, there were six IMD related deaths, five due to serogroup B and one due to serogroup Y. Five of the deaths were in children <5 years of age and one was in a young adult.

Discussion

The epidemiology of IMD in Ireland has remained largely unchanged over the last three years, with incidence rates now almost a third of what they were in 1999/2000.

The greatest reduction has been in serogroup C disease with incidence rates declining from 3.7 per 100,000 in 1999 to 0.1 per 100,000 in 2006, a reduction of 97%. This is a reflection of the huge impact the introduction the MenC vaccine has had on the incidence of IMD in Ireland. The attack rate due to serogroup B disease has halved in recent years, from incidence rates of 8.1 per 100,000 in 1999 to 4.0 per 100,000 in both 2005 and 2006. Serogroup B now accounts for 80% of cases of IMD in Ireland, with well over half of these occurring in young children.

Despite the relative reduction in IMD over the past number of years, the severity, the high mortality rate and the serious adverse sequelae associated with this disease ensure it is treated as a serious public health concern. Effective vaccination is necessary for the comprehensive prevention and control of meningococcal disease. However, meningococcal vaccines available are only effective against serogroups A, C, W135 and Y. For several reasons conventional methods of vaccine development have failed to produce a vaccine with a broad range of protection against circulating serogroup B strains. The incidence of *N. meningitidis* serogroup B is highest amongst infants and the development of a serogroup B vaccine that is effective in this age group is a priority. Until such time an effective MenB vaccine is available, IMD will remain a significant cause of morbidity and mortality in children and young adults in Ireland.

Margaret Fitzgerald and Suzanne Cotter, HPSC

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Staphylococcal Scalded Skin Syndrome Cluster in HSE Mid-West

A cluster of five cases of staphylococcal scalded skin sydrome (SSSS) occurred in the HSE Mid-West area between February and May 2007. All cases were caused by methicillin sensitive *Staphylococus aureus* (MSSA). Pulsed-field gel electrophoreisis (PFGE) typing found three of the four isolates typed to date to be indistinguishable. Microbiological investigations are ongoing.

The five cases occurred in full-term babies born at the Mid-Western Regional Maternity Hospital, Limerick. All neonates were well going home and presented to the paediatric department at the Mid-Western Regional Hospital between two and nine days after discharge. All have made a complete recovery.

Control measures

Control measures included heightening of infection control measures in the Mid-Western Regional Maternity Hospital and use of chlorhexidine powder for umbilical care and also an anti-staphylococcal wash. Mothers were given an SSSS advice leaflet on discharge. All general practitioners, and 'Shannondoc' (the local general practitioner out-of-hours service), were advised of the cluster and asked to be vigilant regarding soft tissue infections. They were also advised to take swabs from any suspect cases and provide a prescription for oral flucloxacillin. Directors of public health nursing were also advised of the situation.

All medical, nursing and nursing attendant staff are being screened for *S. aureus* carriage. Healthcare workers found to have *S. aureus* [both MSSA and methicillin-resistant *S. aureus* (MRSA)] nasal carriage are being treated with mupirocin and those with dermatologocal lesions with chlorhexidine scrub. Screening is being repeated following treatment. To date 33% of those screened have *S. aureus* nasal carriage.

Background

SSSS is a blistering skin condition which looks like a burn or a scald. It is caused by exfoliative toxin producing strains of *S. aureus*. At least two exfoliative toxins A and B (ETA and ETB) can cause disease.[1,2]

Most cases occur in infants and young children.[3] In neonates, onset typically occurs between three and sixteen days.[2] The higher incidence of generalised SSSS in children may be due to less-efficient renal clearance of the toxin and immunological immaturity.[3] SSSS is rare in adults. Adults with SSSS are most often immunocompromised or have renal failure.[3]

Clinical features

Illness begins with erythema and fever. Bullae form and quickly rupture leaving extensive areas of denuded skin.[2] In neonates, the lesions are mostly found on the perineum and/or peri-umbilically while the extremities are more commonly affected in older children.[1,2] Patients with extensive lesions are susceptible to poor temperature control, extensive fluid loss and secondary infection. They may become septicaemic and present with hypotension, neutropenia, and respiratory distress.[2]

Treatment

Antibiotic treatment with a ß-lactamase resistant penicillin such as flucloxacillin is usually effective.[1] Affected neonates must be isolated with strict barrier nursing.[2] Management of dehydration, hypothermia, pain and secondary infection are particularly important.[2] Steroids are contraindicated.[2]

Prognosis

Fever and erythema usually resolve in 2-3 days. The prognosis in children is good with mortality less than 5%. However, mortality rates in adults are high (60%), usually due to underlying illness.[2]

Outbreaks

Most cases occur sporadically. However, outbreaks in neonatal nurseries have been reported.[4,5,6] Outbreaks of SSSS have been linked to *S aureus* carriage by healthcare workers.[5,6] Nasal carriage of *S. aureus* occurs in 35% of the normal population.[7]

Prevention and control

There is evidence to suggest that the use of antiseptic powder for umbilical care decreases *S. aureus* colonisation.[8] Management of outbreaks in neonatal nurseries has included infection control measures,[4,5,6] use of anti-staphylococcal washes,[4] and screening and treatment of carriers[5,6] as well as exclusion of a healthcare worker with *S. aureus* colonised dermatological lesions.[5]

R Fitzgerald, N O'Connell, R Philip, C MacDonagh-White, HSE Limerick

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Hepatitis B Reactivation in a Dialysis Unit

In April 2005, a case of reactivation of hepatitis B virus (HBV) infection occurred in a patient undergoing haemodialysis in an Irish hospital.[1] This incident potentially affected patients attending hospitals throughout the country, so a national incident team was set up to coordinate the response to the incident.

A total of 306 dialysis patients, attending 17 different dialysis centres (14 in Ireland and three elsewhere in Europe), were identified as having been potentially exposed to HBV as a result of this incident. A programme of HBV serological testing and HBV vaccination was instituted. There was no evidence that any patient acquired HBV infection as a result of cross-infection from the index patient, although 11 patients (3.6%) had evidence of past infection [HBV core antibody positive, HBV surface antigen (HBsAg) negative]. The majority of patients in this cohort (62.7%) were of unknown HBV vaccination status, 13.4% were fully vaccinated, 4.6% partially vaccinated and 15.7% unvaccinated. Of 239 tested for HBV surface antibody titre, 183 (76.6%) had a titre <10mIU/ml. National and international guidelines recommend HBV vaccination for patients with chronic renal failure.[2,3,4]

Local incidents in dialysis units can have national implications due to the frequent patient transfer between units. This incident highlighted serious deficiencies in current structures and practices in Ireland, and a lack of appropriate guidelines. However, there were positive outcomes from this incident. The majority of Irish dialysis patients have now been vaccinated against HBV, and lessons learned have been used to develop national guidelines on HBV vaccination and testing and on the management of incidents of blood-borne viral infections in dialysis units.

More detailed information is available at http://www.eurosurveillance.org/em/v12n04/1204-224.asp.

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