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Cryptosporidiosis Outbreak among Irish Holidaymakers, Spain, July 2003

In July 2003, an outbreak of cryptosporidiosis was reported among British holidaymakers who were guests at a resort in Alcudia, Majorca.[1] To date 391 cases, of which 214 (55%) were laboratory-confirmed, have been reported among English, Welsh, Scottish and Northern Irish tourists who stayed in the same Spanish hotel, in Alcudia.

Following a national alert, 24 Irish cases, from four different health boards, have been reported to the National Disease Surveillance Centre (NDSC). Eleven (46%) have been laboratory-confirmed. Forty five percent of cases were male. Information on age was available for 23 cases. The median age was 13 years, with a range from 5 to 68 years.

The dates of travel and onset of illness suggest that the infections were acquired abroad (incubation period for cryptosporidiosis is generally 1-12 days, median 7 days). The dates of onset of illness were between the 14th and 28th July 2003 (figure 1). The dates of outward-bound travel were between the 11th and 20th July 2003 and of return were between the 25th and 27th July 2003.

SCIEH has reported a small number of cases of cryptosporidiosis among tourists to Majorca who stayed at other hotels.[2] Similarly, NDSC has received a report of one additional Irish cryptospoidiosis case (the red square in the epicurve) who recently travelled to Majorca but did not stay at the hotel linked with the outbreak.

Health authorities in Spain have investigated the incident. *Cryptosporidium* oocysts were reported to have been identified in water backwashed from filters at the hotel swimming pool where many of the cases had been bathing. The pool was subsequently drained and the filters have been refurbished.[1]

Cryptosporidium is a protozoan parasite found in humans, many animals and in birds. Infection does not always give rise to symptoms. In healthy individuals, *Cryptosporidium* is an important cause of self-limiting but profuse watery diarrhoeal illness, with abdominal cramps, nausea, and vomiting. However, in immunocompromised patients, the disease can be more serious.[3]

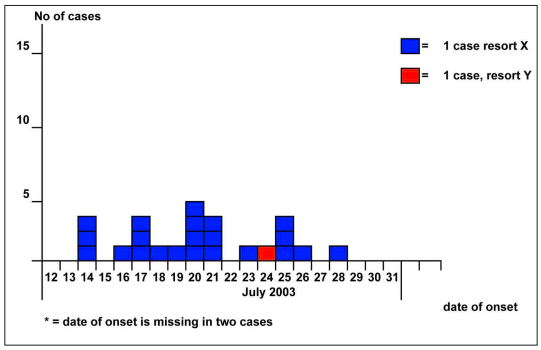

Figure 1. Cases of cryptosporidiosis (n=25) among Irish holidaymakers to Majorca, by date of onset*

Cryptosporidium can be transmitted via water that is contaminated with human or animal faeces. There have been a number of documented outbreaks of *cryptosporidiosis* attributed to potable water, well water, spring water and surface water. Several outbreaks have also been associated with contaminated recreational water in swimming pools. *Cryptosporidium* are a particular risk in swimming pools because they are resistant to chlorine and the filters used in many pools are inefficient at removing oocysts because of their small size.[4]

In Ireland, the national incidence of cryptosporidiosis is unknown. Cryptosporidiosis is notifiable only in infants as 'gastroenteritis under two'. In 2002, of 1717 cases of 'gastroenteritis under two' notified to NDSC, 148 (9%) cases were due to *Cryptosporidium* (provisional data). In 2001, the incidence of cryptosporidiosis in the Western Health Board and in the South Eastern Health Board was reported as 19/100,000 and 18/100,000 population respectively.[5,6]

There is no legislation in Ireland that specifically regulates the use and operation of swimming pools for the purpose of preventing infection. However, civil law still places a duty of care on the operator of a swimming pool to his/her customers. Guidelines in relation to health standards in swimming pools and other recreational pools have been produced. The guidelines cover the structure of pools, water quality and treatment, cleaning, and maintenance and management systems. These guidelines serve as a useful guide to the leisure industry.[7, 8, 9]

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Epidemiology of Verotoxigenic *E. coli* O157 in Ireland, 2002

Verotoxigenic *E. coli* (VTEC) are so-called because of their ability to produce one or both of two verotoxins (VT1 and VT2). They cause a wide range of illnesses, from mild diarrhoea to haemorrhagic colitis with severe abdominal pain and bloody diarrhoea. Illness is usually self-limiting and resolves after about eight days. However, 2-7% of cases develop haemolytic uraemic syndrome (HUS), a form of renal failure; this is a more likely complication in young children. In fact, VTEC are the most common cause of diarrhoea-associated HUS in children.[1] In adults, VTEC infection may be followed by thrombotic thrombocytopaenic purpura (TTP).

The primary reservoir is cattle, although VTEC have been isolated from a variety of healthy animal carriers including sheep, horses, goats and wild birds. While this organism was first recognized as a foodborne pathogen (the 'burger bug'), it is now known that it can also be transmitted through water, the environment and by direct contact with infected animals. Person-to-person spread has also been documented.

E. coli O157 is the most commonly reported VTEC in Ireland, the UK and the US, although other serogroups are capable of causing the same spectrum of illness, including O26, O111, O103 and O145. Many cases of O26 have been reported in Ireland, including several cases in children during an outbreak in 1999.[2]

Facilities for VTEC diagnosis and confirmation in Ireland have improved greatly over the last few years. Since October 2000, the Public Health Laboratory at Cherry Orchard Hospital, Dublin has provided an *E. coli* O157 and non-O157 diagnostic service for clinical and food samples, including *E. coli* serotyping and verotoxin detection.

Methods

This is the fourth year that NDSC, in co-operation with Directors of Public Health in each health board region, have operated the epidemiological surveillance system for VTEC O157. Since 1999, specialists in public health medicine, senior area medical officers, area medical officers, microbiologists, medical scientists, surveillance scientists, infection control nurses, principal environmental health officers and environmental health officers participate in a system whereby a standard dataset of information is collected at health board level on each case identified and reported to NDSC. This information includes socio-demographic data, clinical data, possible risk factors and information on links between cases. Some participants in the system also notify non-O157 VTEC. The case definitions that have been used in this system are as follows:

- *Suspected:* a case of post-diarrhoeal HUS or TTP.
- *Probable:* a case with isolation of *E. coli* O157 from a clinical specimen (asymptomatic or symptomatic), pending confirmation of H7 or Shiga toxin or a clinically compatible case that is epidemiologically linked to a confirmed or probable case.
- *Confirmed:* a case that has isolation of *E. coli* O157:H7 from a specimen or isolation of Shiga toxin-producing *E. coli* O157:NM (non-motile) from a clinical specimen.

Probable cases that are subsequently confirmed as not H7 or Shiga toxin-producing are removed from the database. A travel-associated case is defined as one where there has been international travel within two weeks prior to onset of illness.

Results

Seventy confirmed cases of VTEC O157 were notified to NDSC that had a date of onset of symptoms during 2002. Two of these cases occurred in non-residents and are not therefore included in the estimation of population-based rates. However, they are included in the descriptive epidemiology. The numbers of confirmed cases and the crude incidence rates of VTEC O157 in Ireland from 1996-2002 are shown in table 1.

Table 1. Number of cases of confirmed VTEC O157 and crude incidence rate [95% confidence interval (CI)] in Ireland, 1996-2002

Year	Numbers of confirmed cases (number confirmed cases including non-residents)	Crude incidence rate[a] (95% CI) per 100,000 population
1996[b]	8	0.2 (0.1-0.4)
1997[b]	31	0.8 (0.5-1.2)
1998[b]	76	2.1 (1.6-2.6)
1999	51	1.4 (1.0-1.8)
2000	37(42)	0.9 (0.6-1.3)
2001	50 (52)	1.3 (0.9-1.6)
2002	68 (70)	1.7 (1.3-2.2)

[a] For the years 1996-1999, census figures for 1996 were used while figures from the 2002 census were used to calculate rates from 2000-2002. The latter rates consequently differ from those published previously.

[b] Data for the years 1996-1998 were taken from the report of the FSAI VTEC Working Group.[3]

Suspect/probable cases

An additional 9 suspect/probable cases were reported to NDSC in 2002 that were not subsequently confirmed. These included 4 from the Mid-Western Health Board, three from the Southern Health Board and one each from the Western and North-Western Health Boards. Four of these cases had HUS. The remaining 5 cases were symptomatic contacts of confirmed cases; one of these was found positive by serodiagnosis. However, as these cases do not fit the case definition for confirmed cases, they are not included in any of the following analyses.

Regional distribution

As in previous years, regional variation was noted in the numbers of cases reported (table 2), with the highest rates in the Midland, Western and South-Eastern Health Boards. These 3 health board regions appear to have consistently higher rates of VTEC O157 infection.

Table 2. Crude incidence rate (CIR) and age standardised incidence rate (ASIR) with 95% confidence intervals of confirmed cases of VTEC O157 by health board of residence, Ireland, 2002

Health Board	2002	
	CIR (95% CI) per 100,000	ASIR (95% CI) per 100,000
ERHA	0.9 (0.4-1.3)	0.9 (0.4-1.4)
MHB	6.7 (3.3-10.0)	6.5 (3.2-9.8)
MWHB	0.3 (0.3-0.9)	0.3 (0.3-0.9)
NEHB	0.3 (0.3-0.9)	0.3 (0.3-0.8)
NWHB	1.4 (0.2-2.8)	1.4 (0.2-3.0)
SEHB	3.5 (1.8-5.3)	3.5 (1.7-5.3)
SHB	0.5 (0.1-1.1)	0.5 (0.1-1.1)
WHB	4.7 (2.6-6.9)	4.8 (2.6-7.0)
Total	**1.7 (1.3-2.2)**	

Age-sex distribution

The highest incidence rate was recorded for young children, a trend also noted over the last few years (figure 1).

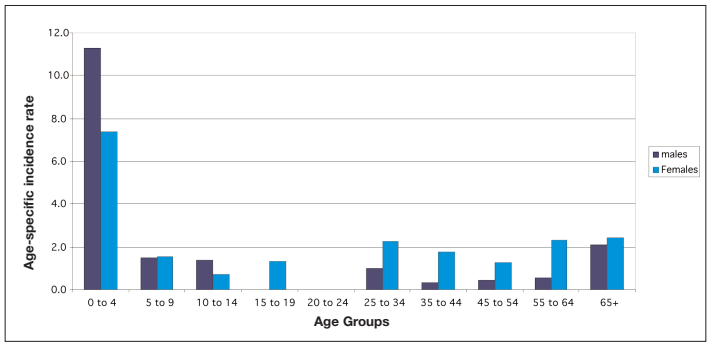

Figure 1. Age-specific incidence rate (per 100,000 population) of confirmed cases of VTEC O157, Ireland, 2002.

Clinical features

In total, 61 out of the 70 confirmed cases (87%) were symptomatic. Reported symptoms included: bloody diarrhoea in 31 cases (51%), and HUS in 5 cases (8%). Of the 5 cases of HUS, 4 occurred in children under 5 years of age. One person who was admitted to hospital with VTEC O157 in 2002 developed additional medical problems while hospitalised and died a number of weeks later. It is unclear to what extent the VTEC infection contributed to the precipitation of their subsequent medical problems.

Seasonality of VTEC O157 cases

The majority of cases in 2002 occurred in late summer/early autumn with a peak in August (figure 2).

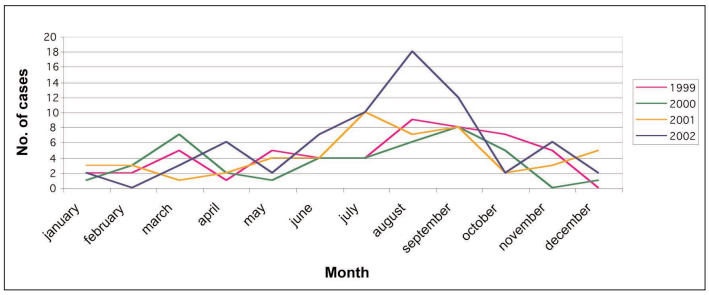

Figure 2. Confirmed cases of VTEC O157 by month of onset of symptoms, Ireland, 1999-2002

Travel-association

Nine cases of VTEC O157 were travel-associated. The countries visited within 14 days of onset of illness were Canada (1), UK (1), France (1), Spain (4), Canary Islands (1) and Tunisia (1).

Microbiological investigation

In 2002, phage-typing results were available for 54 cases out of 70. In total seven different phage types were reported. As in previous years, the predominant type detected was PT 32 (n=30). This was followed in frequency by PT 8 (n=9), PT 21/28 (n=6) and PT 14 (n=4), all of which were previously reported in Ireland.

Epidemiological investigation

As a result of following up apparently sporadic cases in 2002, 14 family outbreaks were detected by health board personnel among 29 confirmed cases, demonstrating the importance of investigating each case of VTEC infection. No general outbreaks were reported and no links were found with any food or water source.

Risk exposures

Descriptive epidemiological information was collected on all reported cases in an attempt to identify potential risk factors for exposure to VTEC. Seven (10%) cases reported consumption of unpasteurised milk or cheese. Of 58 cases where information was collected on water source, the water supply was public in 34 (59%) cases, private well water in 12 (21%) cases, from a group scheme in 6 (10%) cases and recorded as other (not public and not well) in 6 (10%) cases. Contact with farm animals was reported in 25 (36%) cases.

Non-O157 VTEC

In 2002, one confirmed case of VTEC O26 was reported to the enhanced surveillance system.

Discussion

Seventy confirmed cases of VTEC O157 infection (1.7 per 100,000 population) were reported in Ireland in 2002, the highest rate recorded since 1998. Irish incidence rates are comparable to those published for Northern Ireland in 2002 (1.6/100,000).[4] Higher rates have consistently been reported for Scotland (4.5/100,000 in 2002).[5]

Fifty-seven per cent of cases had a date of onset between July and September. While a higher incidence during this time is a feature of VTEC infection, the particularly high rate in the summer of 2002 was influenced by the reporting of 14 cases of VTEC O157 in the Midland Health Board region with a date of onset during these 3 months. Two family outbreaks made up half of these cases, while the remaining 7 cases had 3 different phage types. Certain parts of the country appear to have consistently higher rates of VTEC O157 infection. The Midland Health Board, along with the Western and South-Eastern Health Boards, reported the highest rates of infection in 2000, 2001 and 2002.

Nine HUS cases were reported to the surveillance system in 2002, but only 5 were confirmed as being caused by *E. coli* O157. The remaining 4 suspect cases were children under 7 years of age; 2 were male and 2 were female. Three of the four also suffered from bloody diarrhoea. In HUS surveillance studies in different countries, up to 91% of cases were found to have evidence of VTEC infection.[1] It is possible that some or all of these 4 suspect cases were caused by VTEC.

An additional probable case in the database was epidemiologically linked to a confirmed case but was confirmed only by serodiagnosis. The current case definition requires cases to be culture positive. While this case is a laboratory-confirmed case, it is not included in our analyses because it falls outside the case definition. Under the new case definition in the proposed infectious disease legislation, cases of HUS and TTP which are serodiagnosed as being caused by VTEC, will be included.

The reporting of a VTEC O26 case again in 2002, highlights the importance of extending the enhanced surveillance system to non-O157 VTEC. This was probably not the only confirmed case of O26 in Ireland, in 2002. Additional O26 cases were notified under the weekly notification system, although their toxin-producing abilities are not reported.

Dr. Patricia Garvey, Dr Barbara Foley and Dr Paul McKeown, NDSC

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The Emergence of West Nile Virus in North America

A cluster of older, previously healthy people with fever, altered mental status and profound muscle weakness appearing in New York City in the late summer of 1999 generated considerable interest and then shock when it was realised that the causative pathogen was West Nile Virus (WNV), an Old World flavivirus, never previously described in North America.[1] The vector responsible for transmission of WNV is the mosquitoe; its natural hosts are birds.

The initial outbreak produced 62 cases of meningoencephalitis and 7 deaths. Over the following two seasons, the numbers of ill and dead as a result of WNV infection remained largely static. It appeared as if the US was playing host to yet another minor outbreak of arthropod-borne viral encephalitidis, much less of a problem, in public health terms, than the two best known of North Americas indigenous examples; LaCross encephalitis and St Louis encephalitis.

This all changed in 2002. Continued expansion of the virus produced an arboviral outbreak on a scale unprecedented in North America. In 2002, there were 4156 probable or confirmed human cases of WNV-associated illness and 284 deaths across 40 states.[2] As of 18 August 2003, there have been 536 cases and 11 deaths reported.[3] In Canada, over 360 human cases of WNV had been identified with 11 deaths for 2002.[4]

Speculation as to the origin of the initial outbreak has produced a wide variety of theories, the most likely being that the origin of the initial New York City outbreak was a variant virus from Israel, possibly imported inadvertently on board an aircraft.[5]

WNV was first isolated from the serum of a febrile female patient in the West Nile district of Uganda, in 1937.[6] Subsequent outbreaks of the disease occurred infrequently. Large epidemics occurred in Israel in the early 1950s and in South Africa in 1974.[7] Since the mid-1990s, worrying epidemiological features in WNV have become apparent, including increasing frequency of outbreaks involving humans, an apparent increase in more severe human disease and high avian death rates accompanying outbreaks of human disease in Israel and the US.[8]

In Europe, WNV is commonly encountered in southeastern Europe and Russia. The largest European outbreak was in Romania in 1996, with more than 500 clinical cases and a case fatality rate approaching 10%.[9] In Western Europe, WNV infection has been restricted to mosquitoes, birds, horses and occasional mild human cases, principally around the Rhone delta.[10]

During 2002, unexpected and worrying methods of transmission of WNV were noted during the US outbreak.[11] These included 61 possible cases of transfusion transmitted WNV infection,[12] as well as a number of cases that were associated with organ transplantation.[13] Breastfeeding, transplacental transmission and occupational exposure have all been implicated methods of transmission, albeit in very small numbers.[14]

The spectrum of illness produced by infection with WNV is similar to other mosquito-borne arboviral encephalitides:

a) *Asymptomatic:* Following exposure, about 80% of subjects will be asymptomatic.
b) *Minor Illness:* Another 20% will develop a mild influenza-like illness lasting 3 to 6 days, with fever, headache and generalised aches and pains (known as West Nile Fever).
c) *Severe Illness:* A small proportion of infected people (less than 1%) will go on to develop more severe disease.

- Case fatality rates during recent outbreaks have ranged from 10% in Romania (1996) to 12% in New York (1999) and 14% in Israel (2000).
- The most significant risk factor for developing severe neurological disease is older age (especially over 50; those over 80 are particularly vulnerable).
- Encephalitis is more commonly reported than meningitis.
- In recent outbreaks, symptoms occurring among patients hospitalised with severe disease include pyrexia, profound muscle weakness, headache, altered mental status and gastrointestinal symptoms.
- A minority of patients with severe disease developed a maculopapular or morbilliform rash involving the neck, trunk, arms, or legs.
- About 10% of patients demonstrated complete flaccid paralysis (initially, the first cases were thought to be suffering from Guillain-Barré syndrome).
- Neurological presentations included: ataxia and extrapyramidal signs, cranial nerve symptoms, optic neuritis and seizures.
- Encephalopathy along with severe muscle weakness, changes in level of consciousness and advanced age were the most powerful clinical predictors of death in those with severe disease.

As there is a potential risk of exposure of Irish citizens to WNV, the National Disease Surveillance Centre has issued advice for travellers to North America that can be found at www.ndsc.ie.

Dr Paul McKeown, NDSC

References

References are available on request.

Salmonella Monthly Report (July 2003):

Strains are allocated to months based on the date of receipt of the isolate from the referring laboratory. These figures are provisional as work may not be finished on particular strains at the time of publication. Data are provided courtesy of Prof Martin Cormican and Dr Geraldine Corbett-Feeney, INSRL.

Health Board	E	M	MW	NE	NW	SE	S	W	Total
S.Dublin	0	0	0	1	0	0	0	0	1
S.Enteritidis	12	4	1	4	1	2	4	6	34
S.Hadar	0	0	0	0	1	0	0	0	1
S.Java	0	0	0	0	0	0	1	0	1
S.Kottbus	1	0	0	0	0	0	0	0	1
S.Muenchen	1	0	0	0	0	0	0	0	1
S.Newport	1	0	0	0	0	0	0	0	1
Paratyphi A	0	0	0	0	0	0	0	1	1
S.Poona	1	0	0	0	0	0	0	0	1
S.Sandiego	1	0	0	0	0	0	0	0	1
S.Typhimurium	4	0	2	2	0	1	0	0	9
S.Virchow	0	0	0	0	0	0	0	1	1
S.Weltevreden	1	0	0	0	0	0	0	0	1
Unnamed	0	0	0	0	1	0	0	1	2
Total	**22**	**4**	**3**	**7**	**3**	**3**	**5**	**9**	**56**