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Avian Influenza

Poultry/Bird Outbreaks

The current outbreaks of avian influenza started in South East Asia in mid-December 2003 and have now spread to parts of Europe, the Middle East, Africa and India. The outbreaks are the largest and most severe on record. Never before in the history of this disease have so many countries been simultaneously affected, resulting in the loss of so many birds. Further spread to new areas is expected.

It is not clear what role migratory birds have in the spread of the current outbreaks. It had been thought that wild waterfowl introduce avian influenza viruses in their low pathogenic form to poultry flocks but do not carry or directly spread highly pathogenic viruses. However, scientists are increasingly convinced that at least some migratory waterfowl are now carrying the H5N1 virus in its highly pathogenic form, sometimes over long distances, and introducing the virus to poultry flocks and other birds in areas that lie along their migratory routes. Should this new role of migratory birds be scientifically confirmed, it will mark a change in a long-standing stable relationship between the H5N1 virus and its natural wild-bird reservoir. The movement of poultry and poultry products is also believed to be an important mode of dissemination of the virus within countries.

Table 1. The number of confirmed human cases and deaths due to avian influenza H5N1 in the current outbreaks up to 27 February 2006

Country	No. of cases	No. of deaths	Date of first case	Date of last case
Vietnam	93	42	Jan 04	Nov 05
Thailand	22	14	Mar 04	Dec 05
Cambodia	4	4	Feb 05	Apr 05
Indonesia	27	20	Jul 05	Feb 06
China	14	8	Nov 05	Feb 06
Turkey	12	4	Jan 06	Jan 06
Iraq	1	1	Jan 06	Feb 06
Total	**173**	**93**		

Countries affected by the current outbreaks include: Republic of Korea, Vietnam, Japan, Thailand, Cambodia, Lao PDR, Indonesia, China (including Tibet), Malaysia, Russia, Kazakhstan, Mongolia, Turkey, Romania, Croatia, Ukraine, Iraq, Nigeria, Azerbaijan, Bulgaria, Greece, Italy, Slovenia, Iran, Austria, Germany, India, Egypt, France and Hungry. Japan, Republic of Korea, and Malaysia had announced control of their poultry outbreaks and were considered free of the disease. However, Malaysia has recently reported a fresh outbreak after being free of disease for more than one year.

Human Outbreaks

As of 27 February there have been 173 confirmed cases of human avian influenza A/H5N1 reported to WHO and 93 deaths. The countries affected include Vietnam, Thailand, Cambodia, Indonesia, China, Turkey and Iraq (table 1).

Discussion

H5N1 is spreading rapidly across the world. This increases the opportunity for the virus to mutate and allow it to transmit easily from person to person. If this happens it could trigger an influenza pandemic. The Director-General of WHO has indicated that the highest priority should be given to warning people about the dangers of close contact with sick or dead birds infected with H5N1. The slaughtering, defeathering, butchering, or consumption of infected poultry are high risk behaviours. Experience in Asian countries and Turkey has shown that immediate, clear public information is critical to the protection of human health.

All the above information is available on the WHO website at www.who.int/en/.

New Case of Transfusion-associated vCJD in the UK

A third case of variant Creutzfeldt-Jakob disease (vCJD), in a patient who received a blood transfusion from a donor who later developed vCJD, has recently been diagnosed in the UK. All three cases received a transfusion with non-leucodepleted red blood cells and each received blood from different donors. Since October 1999, leucocytes have been removed from all blood used for transfusion in the UK. However, the effect of leucodepletion on the reduction of the risk of transmission of vCJD from an infected donor is uncertain.[1]

A national study is being undertaken in the UK since 1997 to collect evidence about transmission of CJD and vCJD via the blood supply. To date, 160 cases of vCJD have been identified in the UK. Review of data at blood centres has found records for 23 of the 160 vCJD cases (prior to their vCJD diagnosis). For 18 of these 23 cases, blood components were issued to hospitals for transfusion, and 66 recipients of these vCJD-implicated blood donations have been identified. Many of these recipients have died and the remaining have been informed of their potential exposure to vCJD by blood transfusion and have been asked to take certain precautions to reduce the risk of onward person-to-person transmission of vCJD during medical procedures.[1]

Four cases of vCJD have been reported in Ireland to date. None of these have been reported as being related to receipt of blood or blood products. Leucocytes have been removed from all blood used for transfusion in Ireland since November 1999.

Reference

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Campylobacteriosis in Ireland, 2004

Background

Campylobacteriosis is the commonest reported bacterial cause of infectious intestinal disease in Ireland. Two species account for the majority of infections: *C. jejuni* and *C. coli*. Illness is characterised by severe diarrhoea and abdominal pain. Symptoms may subside after a number of days or may persist for weeks. Rarely, more severe sequelae may develop such as reactive arthritis, Reiter's syndrome, or HUS. Approximately 1 in every 1,000 cases leads to a severe neurological disorder called Guillain-Barré Syndrome (GBS). Rehydration and electrolyte replacement are the cornerstone of treatment. Antibiotics are indicated in cases of severe or prolonged illness. Undercooked meat especially poultry is often associated with illness as is unpasteurised milk and untreated water. However, the risk factors for the majority of infections remain unknown.

Methods

Human campylobacter infection became a statutorily notifiable disease for the first time on 1 January 2004 under the Infectious Diseases (Amendment) (No. 3) Regulations 2003.[1] Data for this report were extracted and analysed from the CIDR system (Computerised Infectious Disease Reporting System). Data from 1999 to 2003 inclusive were collected under the Zoonoses Directive as part of the national survey on campylobacter infection in Ireland.

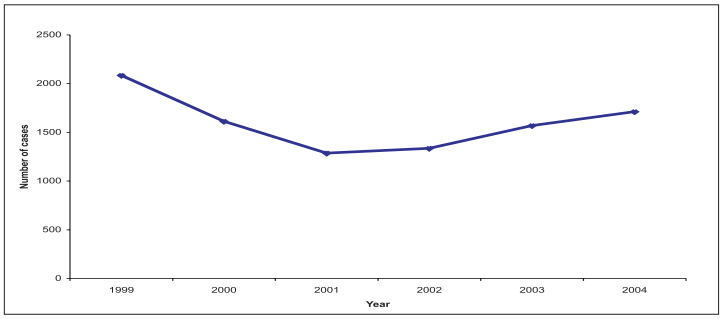

Figure 1. Annual number of cases of campylobacteriosis in Ireland, 1999-2004 (2004 data from CIDR)

Table 1. Number of cases and CIR per 100,000 population of human campylobacteriosis in Ireland by health board, 2004

Health Board	No. of cases	CIR - (incl. 95% C.I.)
ERHA	591	42.2 [38.8 - 45.6]
MHB	134	59.5 [49.4 - 69.6]
MWHB	107	31.5 [25.5 - 37.5]
NEHB	113	32.8 [26.8 - 38.8]
NWHB	92	41.5 [33.0 - 50.0]
SEHB	194	45.8 [39.4 - 52.2]
SHB	240	41.4 [36.2 - 46.6]
WHB	240	63.1 [55.1 - 71.1]
Total	1,711	43.7 [41.6 - 45.8]

Results

Incidence

In 2004, 1,711 notifications of human campylobacteriosis were notified in Ireland. This gives a crude incidence rate (CIR) of 43.7 cases per 100,000 population (table 1). This compared with a CIR of 39.9 cases per 100,000 in 2003. The annual number of cases by year since 1999 is shown in figure 1.

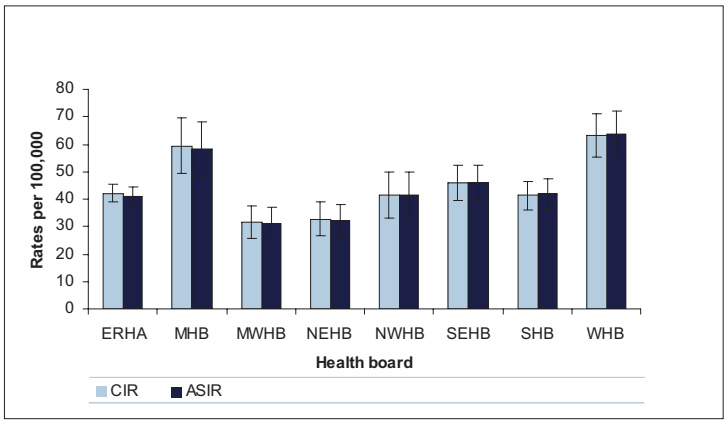

Figure 2. Age standardised incidence rates (ASIR) of human campylobacteriosis in Ireland, and crude incidence rates (CIR) in each health board, 2004.

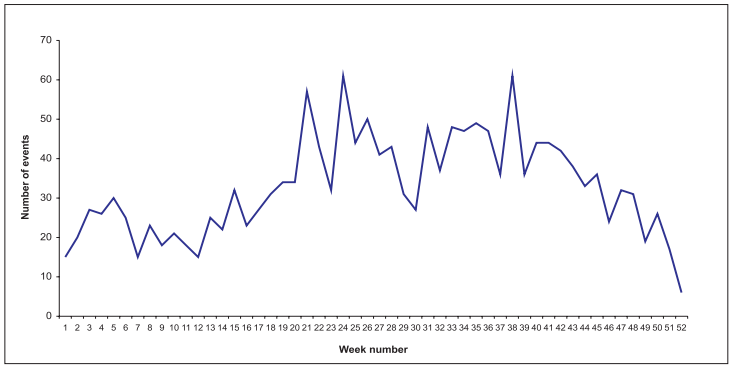

Figure 3: Total cases of campylobacteriosis events by week, 2004 (data from CIDR)

Age standardised rates were calculated to allow comparisons to be made between health board regions without the confounding effects of age (figure 2). In 2004, the highest incidence was reported from the Western Health Board followed by the Midland Health Board. The lowest rate was reported from the Mid-Western Health Board.

Seasonal distribution

Analysis of the data by week of notification is shown in figure 3. A peak in cases is evident in week 24 and again in week 38.

Age

When the distribution of cases for each age group is examined, it is evident that by far the highest burden of illness is seen in children less than five years (figure 4).

Gender distribution

The variation in gender distribution that has been noted since 1999

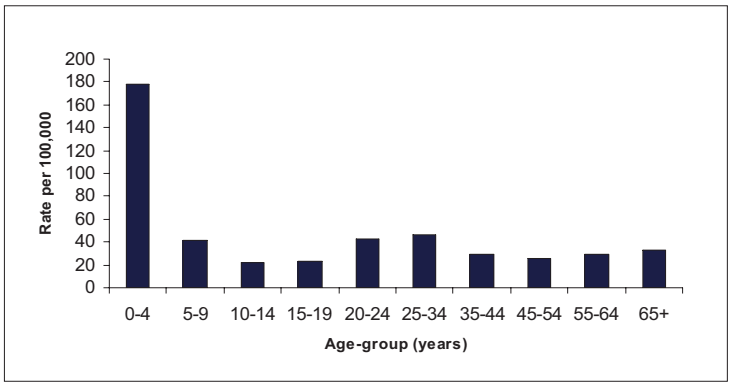

Figure 4: Age-specific incidence rates for campylobacteriosis in Ireland, 2004 (data from CIDR)

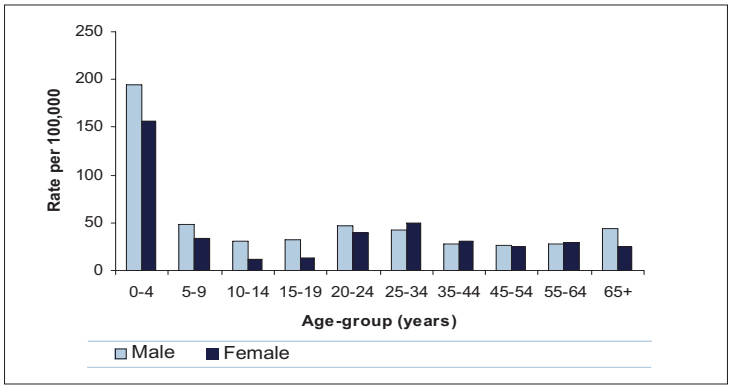

Figure 5. Age-gender adjusted incidence of campylobacteriosis according to age-group in 2004

Table 2. Gender distribution of campylobacter cases by health board, 2004

Health Board	Female	Male	Unknown	Total
ERHA	279	311	1	591
MHB	71	63	0	134
MWHB	45	62	0	107
NEHB	42	70	1	113
NWHB	42	49	1	92
SEHB	84	110	0	194
SHB	110	128	2	240
WHB	105	133	2	240
Total	778	926	7	1,711

was again evident from analysis of the data in 2004, with males accounting for 54.1% of cases and females 45.5% (0.5% unknown) (table 2). This is clearly evident in figure 5 when the data are adjusted for age and sex. In almost all age-groups there is a predominance of male cases.

Outbreak data

There was one small family outbreak of campylobacteriosis involving two persons notified in 2004. The mode of transmission was suspected to be foodborne.

Discussion

In 2004, human campylobacter infections became statutorily notifiable for the first time under the Infectious Diseases (Amendment) (No. 3) Regulations.[1] Therefore in 2004, the data on campylobacteriosis were collated directly from the notifiable disease data on CIDR and not as part of the Zoonoses Directive data collection (as had been the case since 1999).

Analysis of the 2004 infectious disease notification data[2] reveals that campylobacteriosis still remains the most common cause of bacterial gastroenteric infection in Ireland (over four times the number of salmonellosis cases reported in 2004). The CIR of campylobacteriosis increased in Ireland from 39.9/100,000 population in 2003 to 43.7/100,000 in 2004. The 2004 rate was the highest rate reported in Ireland since the year 1999. In most health boards an increase was seen in 2004, especially in the NWHB.

In 2004, a slightly higher rate was noted for Northern Ireland[3] (49.6/100,000), but similar to 2003, much higher rates were observed for England and Wales[4] (79.5/100,000) and Scotland[5] (86.0/100,000) (*provisional data*).

As has been noted since 1999, some interesting epidemiologic features of this pathogen have emerged in recent years. In particular, the higher incidence rates in young children and also in male cases in almost all age-groups. This is a well-reported feature of the illness worldwide.

The first Irish case-control study on campylobacteriosis was conducted in 2004. Its aim was to examine the risk factors that exist for campylobacter infection in the Irish population and help to unravel the aetiology of this disease in Ireland. The study took place in the ERHA region in the ROI and in all four Health and Social Services Boards in NI. The study was completed in 2005. Preliminary findings from the study reveal that eating chicken, and lettuce, and eating out in restaurants/takeaways are major risk factors for campylobacteriosis in Ireland, North and South.[6]

An important conference entitled "*Campylobacter* Surveillance and Research in Ireland – The Way Ahead?" was held in UCD in June 2005. This conference and accompanying workshop, involving international experts, was convened to highlight current knowledge gaps and views on the best way forward for research on campylobacter in Ireland. It is hoped that the findings due to be published this year will prioritise future strategies for campylobacter prevention, control, and surveillance, and help to elucidate some of the complexities of this zoonotic agent.

International research in recent years has indicated that the number of clinically significant *Campylobacter* spp. is being grossly underestimated, with newly emerging strains of *Campylobacter* spp. having an important link to human gastrointestinal illness, and food and environmental samples playing a role in their transmission. The lack of routine typing of clinical isolates in Ireland up until now has limited detailed tracking of these strains through the food chain. A three-year European Commission Research Project entitled 'CampyCheck' involving international collaborators (including Ireland) is aiming to examine ways of improving the recovery and identification of emerging *Campylobacteraceae* in the food and water chain.[7] It is hoped to extend this work to clinical isolates in Ireland in the near future. A recent conference (February 2006) organised by Teagasc (Irish Agriculture and Food Development Authority) presented the findings to date from this project.

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Acknowledgements

We wish to thank all who have provided data for this report, including specialists in public health medicine, senior/medical officers, surveillance scientists, clinical microbiologists, medical scientists, infection control nurses, principal/environmental health officers.

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Increase in Invasive Group A Streptococcal Infection in Ireland?

Background

Group A streptococcus (*Streptococcus pyogenes* - GAS) is the most common bacterial cause of pharyngitis and is the only cause of pharyngitis for which antimicrobial therapy is clearly indicated. GAS can also cause a range of diseases including soft tissue infection, scarlet fever, rheumatic fever, and post streptococcal glomerulonephritis.

Invasive GAS infection (iGAS) is an infrequent complication of GAS infection which carries a significant mortality and requires a prompt diagnosis. There has been concern for some time that the incidence of iGAS is increasing in Ireland. Six cases of iGAS, two of whom have died, have been reported to the HPSC since 1 January 2006. The iGAS sub-committee of the Scientific Advisory Committee of HPSC is in the process of completing Irish guidelines for surveillance and management of iGAS. This document has been sent for consultation and is due to be published in Summer 2006. In the light of these developments, this article highlights the spectrum of iGAS and the appropriate management of invasive infection.

Clinical Presentation

Initial signs and symptoms of iGAS may be non-specific. Clinicians should have a high index of suspicion especially in 'at-risk' patients including young children, those over 65 years, patients with skin trauma or concomitant varicella, young adults exposed to children, immunocompromised patients (e.g. underlying malignancy, HIV infection and high-dose steroid use), diabetes mellitus, underlying heart or chronic lung disease, alcohol abuse, intravenous drug abusers and pregnant women.

IGAS presents in a variety of ways including:

- **Cellulitis**
- **Necrotising fasciitis** (NF) a deep-seated infection of subcutaneous tissue that results in the rapidly progressive destruction of fat and fascia but may spare skin and muscle. Clinical findings are more prominent in the latter stages and include pain and tenderness out of proportion to the appearance of the area and marked systemic symptoms, with early onset of shock and organ failure.
- **Myonecrosis** resulting from haematogenous seeding to muscle usually occurs in association with NF as an expansion of the destructive process, but can be isolated. Early signs include severe pain, swelling and erythema, although muscle compartment syndromes may develop rapidly.
- **Streptococcal Toxic Shock Syndrome** (STSS) characterised by hypotensive shock and multi-organ failure. Fever, and severe pain which is abrupt in onset, are the most common initial symptoms. Twenty percent of patients have an influenza-like syndrome and the majority have clinical signs of soft tissue infection such as localised swelling and erythema which in 70% of cases progress to NF or myositis and requires surgical debridement. Approximately 50% of patients have normal blood pressure (systolic pressure >110 mm Hg) on admission but develop hypotension within the subsequent four hours.
- **Focal iGAS** including meningitis, pneumonia, peritonitis, puerperal sepsis, osteomyelitis, septic arthritis and surgical wound infections.

Case Definition

The case definition of iGAS comprises both clinical and laboratory criteria:

Clinical criteria

IGAS comprises an acute febrile illness that may be associated with STSS. STSS is characterised by hypotension (fifth percentile of systolic blood pressure in children, or less than 90 mmHg systolic pressure in adolescents and adults) and two or more of the following:

- Renal impairment (creatinine greater than twice the upper limit of normal for age)
- Coagulopathy (platelets < 100,000 x 10^6/l or evidence of disseminated intravascular coagulation)
- Liver dysfunction (ALT, AST or bilirubin more than twice the upper limit of normal for age)
- Adult respiratory distress syndrome (pulmonary infiltrates and hypoxaemia without cardiac failure or generalised oedema)
- Generalised erythematous rash that may desquamate
- Soft tissue necrosis (necrotising fasciitis, myositis, gangrene).

Laboratory criteria

Isolation of group A streptococcus (*S. pyogenes*) from a normally sterile site (e.g. blood, cerebrospinal fluid, pleural fluid).

For probable case (STSS only): Isolation of group A streptococcus from a non-sterile site (e.g. throat, sputum, vagina).

A confirmed iGAS case is one that is laboratory confirmed. A probable iGAS case is one that is clinically compatible and meets the probable laboratory criteria.

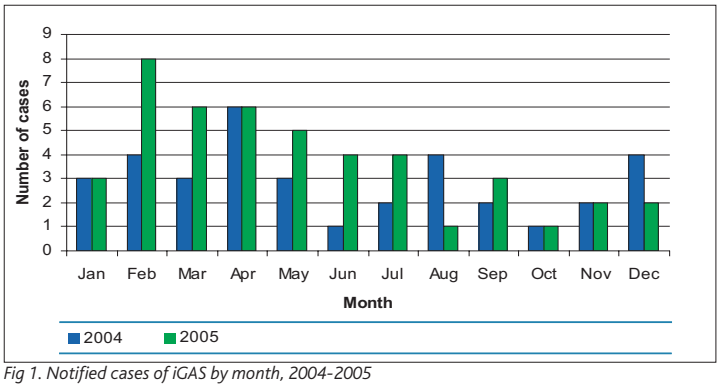

Fig 1. Notified cases of iGAS by month, 2004-2005
Source: HPSC data and CIDR 2006

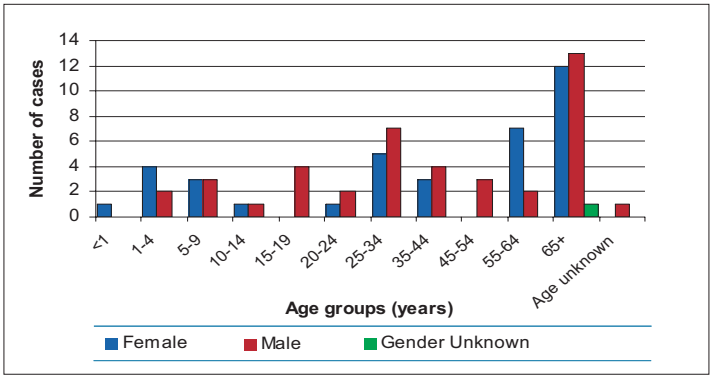

Fig 2. Distribution of iGAS cases by sex and age-group
Source: HPSC data and CIDR 2006

Management of iGAS

Management of iGAS includes prompt initiation of fluid resuscitation and intravenous antibiotic therapy. The recommended **initial empiric antibiotic therapy for adults should include benzyl penicillin 1.2-2.4 grams every four hours, flucloxacillin 1-2 g four times daily, and clindamycin 600–900 mg every 8 hours.** Once iGAS is laboratory confirmed flucloxacillin can be discontinued. It is recommended that clinicians consult the British National Formulary for Children for paediatric doses. When NF or myonecrosis is suspected aggressive surgical debridement is extremely important for establishing a diagnosis and removing devitalised tissue.

IGAS cases should be notified to the local director of public health. Chemoprophylaxis is usually not given to close contacts unless they have symptoms suggestive of localised group A streptococcal infection. However, contacts with symptoms suggestive of iGAS should be immediately referred to the Emergency Department for assessment. Other close contacts should receive a group A streptococcal information leaflet (http://www.hpsc.ie/A-Z/Other/GroupAStreptococcalDiseaseGAS/MainBody,1304,en.html) and be advised to seek immediate medical attention if they develop such symptoms. A heightened index of suspicion for iGAS in close contacts should be maintained for 30 days after the diagnosis is made in the index patient.

IGAS in Ireland

IGAS is a notifiable disease in Ireland since 2004. Since then, eighty cases of iGAS have been notified: thirty-five in 2004 and forty-five in 2005 (figure 1).

This translates to the crude-incidence rate of 0.89/100,000 population in 2004 and 1.15/100,000 in 2005. Age and sex breakdown for the two years 2004 and 2005 combined showed that all age-groups were affected with a higher prevalence among the elderly. Thirty-three percent of all infections occurred in the age-group 65 years or over. Overall, slightly more males (53%) were affected than females (46%) (figure 2).

In summary, while not common, iGAS is an important cause of significant morbidity and mortality that requires prompt clinical recognition and appropriate management.

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Acknowledgement

We wish to thank members of the iGAS subcommittee of the Scientific Advisory Committee of HPSC.

References on request.