Ionad Naisiunta Faire Galair

EPI-INSIGHT

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Editorial Comment on the hGISA Article

The appearance of hetero-glycopeptide-intermediate *Staphylococcus aureus* (hGISA) in Irish hospitals is a serious development. Although the clinical significance of hGISA has yet to be fully elucidated there is increasing evidence that such isolates may be associated with treatment failures. More importantly hGISA may be precursors of glycopeptide-intermediate *S. aureus* (GISA), which have been linked to treatment failure. The potential loss of glycopeptides as an effective therapy for MRSA infections would have grave consequences and highlights the importance of the Strategy for the Control of Antilmicrobial Resistance in Ireland (SARI).

Antimicrobial resistance (AMR) in hospitals results in increased morbidity, mortality and financial cost to the healthcare system. Hospital-acquired bloodstream infection (BSI) caused by methicillin-resistance *S. aureus* (MRSA) has been shown to result in a two-fold increase in attributable mortality compared to hospital-acquired BSI caused by methicillin-sensitive *S. aureus* (MSSA), and a three-fold increase in duration of hospitalisation and attributable costs.[1,2] Recent estimates of attributable cost of MRSA infection in hospitalised patients range from 10,000 euro to 36,000 euro per patient.[3] Overall AMR is thought to account for much of the increase in infectious disease-related deaths seen in the United States since 1980 (CDC data).

Control and prevention of AMR in hospitals requires three key components:

1. **Reliable detection and monitoring of AMR through standardised laboratory methods, effective surveillance systems and reference laboratory support.**
2. **Promotion of prudent antibiotic use through prescriber education and multidisciplinary hospital antibiotic stewardship programmes.**
3. **Preventing the spread of pathogens from patient to patient through promotion of hand hygiene and implementation of evidence-based infection control guidelines.**

Some progress towards ensuring these components are present in Irish hospitals has already been made:

1. The recognition of hGISA in Ireland was made possible through participation in the European Antimicrobial Resistance Surveillance System (EARSS) and the support provided by the National MRSA Reference Laboratory (NMRSARL). The recommendation for all Irish diagnostic laboratories to adopt standardised susceptibility testing, appointment of laboratory surveillance scientists (as recommended under the Strategy for the Control of Antimicrobial Resistance in Ireland (SARI)) and the introduction of the Computerised Infectious Disease Reporting (CIDR) system will greatly enhance AMR surveillance.
2. Recommendations on promoting prudent antibiotic use in hospitals have been prepared by the SARI Hospital Antibiotic Stewardship Working Group and have been distributed for consultation.
3. The SARI Infection Control Working Group has produced draft hand hygiene guidelines, which will shortly be sent out for consultation, and is in the process of updating the 1995 national guidelines on control of MRSA. Additional infection control nurses have also been appointed over the past two years.

Overall there is still a shortfall in relevant staffing and infrastructure for the effective control and prevention of AMR in Irish hospitals. The provision of significant regional SARI funding has gone some way to correct this and the current health services reform process should facilitate more effective control of AMR in the future.

Numerous cost-benefit studies have shown that control of AMR is not just cost effective but actually cost saving. Infection control programmes may even be the most cost effective medical 'intervention' in hospitals, when measured in terms of cost per year of life saved.[4] Effective control and prevention of infections caused by antibiotic-resistant pathogens is a costly undertaking, but one which will save lives, free up much needed hospital resources and save money. The alternative is a return to the pre-antibiotic era, with escalating infectious disease mortality, morbidity and costs, as well as an end to much of the modern medical advances that depend on effective prevention and treatment of infection. That is an alternative we must not allow to happen.

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Influenza A/Fujian-like Viruses in Ireland

Influenza A (H3N2) positive specimens from a boarding school outbreak in South County Dublin in mid-September have been characterized as A/Fujian/411/2002. http://www.eurosurveillance.org/ew/2003/031002.asp Three patient samples were sequenced at the National Virus Reference Laboratory and phylogenetic analysis was carried out at Mill Hill Laboratories; all were identical. A/Fujian-like viruses were first detected in very low numbers during the 2002/2003-influenza season in Europe and also in viruses circulating in Australia and New Zealand during July and August 2003 and most recently in Northern Ireland and England. Although the A/Fujian–like viruses are antigenically drifted from the A/Panama-like strain included in the current influenza vaccine, they are still related and there will be some cross protective immunity from the current vaccine. Vaccination is strongly recommended for those over 65, younger people who have chronic illness, such as lung or heart disease, diabetes or a suppressed immune system and also for key healthcare staff and carers. Influenza activity has started earlier this season than previous seasons, with influenza A currently circulating in Ireland. It is, however, too early to predict whether this early activity will be sustained.

Influenza Season 2002/2003

Introduction

Influenza is one of the commonest and oldest diseases known to man. The impact on public health varies depending on the circulating strain of virus and the level of pre-existing immunity in the community each season.[1, 2]

There are three types of influenza virus A, B and C. Influenza C rarely causes human illness. The clinical course of influenza B changes little from year to year and is usually milder than influenza A. Influenza A varies considerably and is responsible for epidemics and pandemics.[3] Influenza A viruses are divided into three subtypes, on the basis of two surface glycoproteins, haemagglutinin (H) and neuraminidase (N). Minor changes in the surface glycoproteins are known as antigenic drift. Antigenic drift occurs between each influenza season, necessitating the annual reformulation of the influenza vaccine, which is based on the current circulating strains.[4] Major changes in the surface glycoproteins occur infrequently and are known as antigenic shift. These result in the emergence of a novel virus that may be capable of causing an influenza pandemic. The Spanish Flu Pandemic of 1918 is acknowledged as the most devastating, resulting in an estimated 20-40 million deaths worldwide.[3 4]

The 2002/2003-influenza season was the third year of influenza surveillance using computerised sentinel general practices in Ireland. The National Disease Surveillance Centre (NDSC) is working in collaboration with the National Virus Reference Laboratory (NVRL) and the Irish College of General Practitioners (ICGP) on this surveillance project. Influenza activity in Ireland was mild during the 2002/2003 influenza season, peaking in February 2003, with influenza B predominating.

Materials and Methods

Clinical data

Thirty-four general practices were recruited to report electronically, on a weekly basis, the number of patients with influenza-like illness (ILI). ILI is defined as the sudden onset of symptoms with a temperature of 38°C or more, with two or more of the following: headache, sore throat, dry cough and myalgia. Patients were those attending for the first time with these symptoms. In total, the 34 sentinel general practices represent 2.4% of the national population. Practices were located in all health boards with their location based on the population of each health board.

Virological data

Sentinel GPs were requested to send a combined nasopharyngeal and throat swab on one patient per week where a clinical diagnosis of ILI was made. Swabs were sent to the NVRL for testing using Shell Vial and PCR techniques and results were reported to NDSC.

Regional influenza activity

The Departments of Public Health sent an influenza activity index (no report, no activity, sporadic-, localised-, regional- or widespread activity) every week, to NDSC. The activity index is analogous to that used by the WHO global influenza surveillance system and the European Influenza Surveillance Scheme (EISS). The index is based on sentinel GP ILI consultation rates, laboratory-confirmed cases of influenza, sentinel hospital admissions data and/or sentinel school absenteeism levels. One sentinel hospital was located in each health board. Sentinel primary and secondary schools in each health board were located in close vicinity to the sentinel GPs.

Weekly influenza surveillance report

NDSC produced a weekly influenza report, which was posted on the NDSC website each Thursday. Results of clinical and virological data were reported, along with a map of influenza activity and a summary of influenza activity worldwide.

Results

Clinical data

GP consultations for ILI were reported on a weekly basis per 100,000 population from week 40, 2002 to week 20, 2003 (figure 1). Influenza activity was very mild during the 2002/2003-influenza season, similar to the 2001/2002 season. The peak GP consultation rate occurred during week 8, with a rate of 52.6 per 100,000 population. This was compared to a peak rate of 29.0 per 100,000 in the 2001/2002 season and 121.0 per 100,000 in the 2000/2001-influenza season. The peak age-specific consultation rate during the 2002/2003 season was in the 10-14 year age group (figure 2). A total of 347 ILI cases were reported by sentinel GPs during the 2002/2003 season.

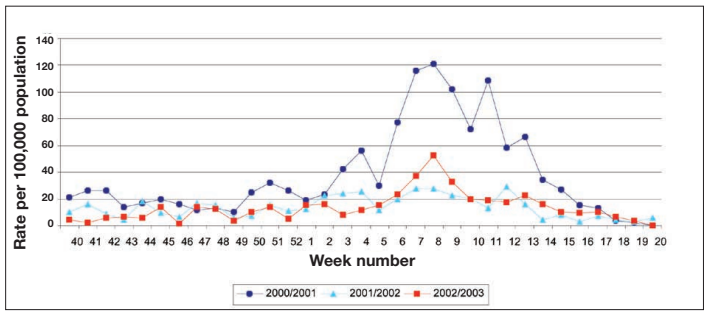

Figure 1. GP consultation rate for ILI per 100,000 population by report week, during the 2000/2001, 2001/2002 and 2002/2003 influenza seasons.

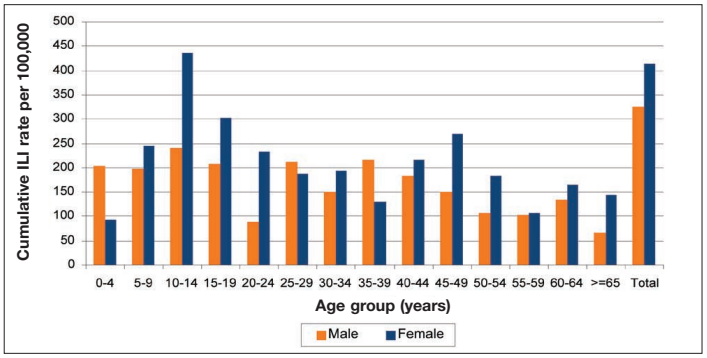

Figure 2. Cumulative age- and sex- specific ILI rate per 100,000 population, week 40, 2002 to week 20, 2003.

The denominator used in the age- and sex- specific consultation rate is from the 2002 census data; this assumes that the age and sex distribution of the sentinel general practices is similar to the national age and sex distribution.

Virological data

The NVRL tested 249 sentinel specimens for influenza virus during the 2002/2003 season, 86 (34.5%) were positive: 27 influenza A and 59 influenza B. Influenza B was the predominant circulating influenza virus type, circulating between weeks 2 and 15, 2003. This was followed by detection of influenza A between weeks 3 and 17, 2003. The highest number of positive swabs detected this season was during weeks 6, 7, and 8 with between 52.4% - 75.0% of swabs positive (figure 3), coinciding with the period of peak clinical activity. Influenza A accounted for 31.4% of positive swabs this season: 6 influenza A (H1) and 21 influenza A (H3N2). Influenza B accounted for 68.6% of positive swabs. Positive influenza virus detections peaked in the 10 - 14 year age group, mainly infected with influenza B.

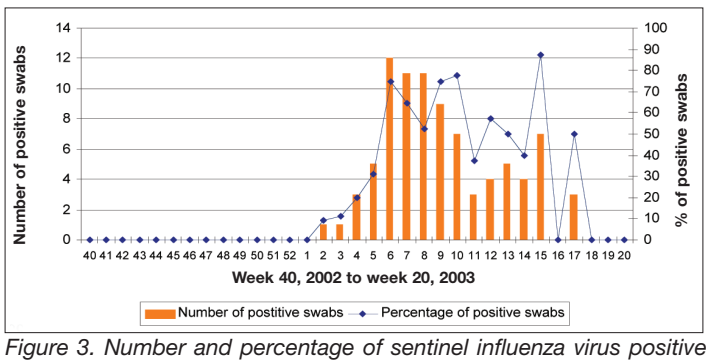

Figure 3. Number and percentage of sentinel influenza virus positive detections during the 2002/2003-influenza season.

Vaccination status and antigenic characterisation

Of the 86 positive influenza virus cases, 64 (74.4%) were not

vaccinated, 2 (2.3%) were vaccinated and 20 (23.3%) were of unknown vaccination status. The NVRL referred one influenza B and 2 influenza A (H3N2) virus isolates to the WHO Laboratory in London for antigenic characterisation. The influenza B virus was antigenically closely related to B/Hong Kong/330/2001-like virus. The 2 influenza A (H3N2) isolates were closely related to A/Panama/2007/99. All isolates were covered by the 2002/2003 influenza vaccine.

Influenza activity by health board/authority

Regional influenza activity peaked between weeks 5 and 13, 2003, with 4 to 7 health boards reporting sporadic activity weekly. During week 8, the period of peak clinical activity, localised influenza activity was reported from the NEHB, with 6 other health boards reporting sporadic activity (figure 4). In some health boards increases in the number of ILI cases were reflected by increases in hospital respiratory admissions and also occasionally by increases in school absenteeism. Between weeks 6 and 8, increased absenteeism was reported in several sentinel primary and secondary schools in the ERHA, NEHB and the SEHB, often associated with ILI.

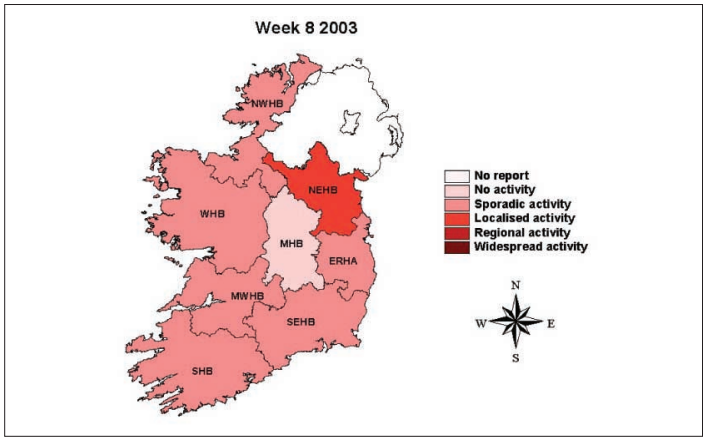

Figure 4. Map of influenza activity by health board, week 8, 2003, the period of peak influenza activity

Influenza activity worldwide

In Northern Ireland, morbidity levels for influenza and ILI were low during the 2002/2003 season, with influenza B predominating.[5] In England, Scotland and Wales, ILI consultation rates peaked in January 2003, with highest consultation rates among children. During late January and early February 2003, influenza B outbreaks were reported in schools in England.[6]

Across Europe, influenza activity was heterogeneous during the 2002/2003 season. Influenza B was the dominant type until week 6, 2003, mainly circulating in the south west and west of Europe. From week 7, 2003, influenza A was the dominant type, mainly circulating in Central Europe. More than 99% of the viruses detected through the EISS have been closely related to the 2002/2003 influenza vaccine strains. A very small number of influenza A (H3N2) viruses (detected in England, Norway, and Switzerland) have, however, shown reduced reactivity to A/Panama/2007/99 antiserum (similar to A/Fujian/411/2002).[7]

In February 2003, outbreaks of highly pathogenic avian influenza, influenza A (H7N7), were reported in several Dutch poultry farms. Following this, avian cases were reported in Belgium and Germany. Human cases of conjunctivitis and ILI, including one death, were associated with the outbreaks. There was also evidence of human-to-human transmission in the Netherlands and Belgium.[8]

In the US, the 2002/2003 influenza season was also mild, peaking in February 2003. Influenza A (H1) and B viruses circulated widely, with the predominant virus varying by region and time of season.[9] In Canada, influenza A (H1N2) was the predominant circulating subtype during the 2002/2003 season; all viruses identified in Canada this season were closely related to the current vaccine strains.[10]

In Hong Kong, influenza activity was mild to moderate during the 2002/2003 season, with influenza A (H3N2) peaking in March 2003.[11] In February 2003, an outbreak of influenza A (H5N1) in Hong Kong was limited to two cases, one of whom died; both cases were members of the same family. The influenza virus that infected these two cases contained no human genes (the virus genes were purely avian in origin); therefore the risk of human-to-human transmission was very low and unlikely to lead to an epidemic. The virus belongs to a different genetic lineage then that of a similar H5N1 virus that caused an outbreak in Hong Kong in 1997, resulting in 18 human cases and six deaths.[12]

The WHO announced the composition of the vaccine for the 2003/2004 Northern Hemisphere influenza season on the 28th of February 2003: A/New Caledonia/20/99 (H1N1)-like virus, A/Moscow/10/99-(H3N2)-like virus (the widely used vaccine strain is A/Panama/2007/99) and B/Hong Kong/330/2001. They recommend that all people in high-risk groups and healthcare workers caring for them be vaccinated as a matter of urgency.[13] This strategy would reduce the burden of influenza and reduce cases of respiratory disease that could be mistaken for SARS or raise suspicion requiring costly investigations.

Discussion

Influenza activity was mild in Ireland during the 2002/2003 influenza season, similar to the 2001/2002 season. This low level of influenza activity was also reflected throughout much of Europe.[7] Influenza B was the predominant virus type circulating this season in Ireland. Influenza activity can be measured not only by GP consultation rates, and laboratory-confirmed cases of influenza but also through school and work absenteeism, hospital admission rates, sales of 'over the counter' medications and deaths.[4] It is of interest to note that the majority of influenza B cases identified in Ireland were aged between 10 and 19 years of age, corresponding with the highest GP consultation rates for ILI. The peak in influenza B detections also coincided with increases in school absenteeism associated with ILI. Increases in the number of ILI cases were also reflected by increases in hospital respiratory admissions in some health boards.

Further expansions and improvements in the present influenza surveillance system are now being introduced for forthcoming seasons, including an increase in the number of sentinel GPs, testing sentinel specimens for respiratory syncytial virus and increasing the number of sentinel swabs. The detection of influenza A (H7N7) in the Netherlands and influenza A (H5N1) in Hong Kong during the 2002/2003-influenza season has emphasised the importance of a timely national surveillance system for influenza.

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Acknowledgements

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Confirmation of Hetero-Glycopeptide-Intermediate *Staphylococcus aureus* among Irish Methicillin-Resistant *S. aureus* Blood Culture Isolates.

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a serious nosocomial problem and therapeutic options may be limited to vancomycin.[1] Reduced susceptibility of *S. aureus* to vancomycin was first reported in 1997 in Japan.[2] Two types of reduced susceptibility to glycopeptide were recognised initially:

- glycopeptide-intermediate *S. aureus* (GISA) where the vancomycin minimum inhibitory concentration (MIC) was 8 mg/L and
- hetero-glycopeptide-intermediate *S. aureus* (hGISA) where the majority of the bacterial population had vancomycin MICs in the susceptible range (≤4 mg/L) but a small minority of bacterial cells (perhaps as few as 1 in 10^6) expressed MICs of 8 mg/L.[3]

The National Committee for Clinical Laboratory Standards (NCCLS) MIC breakpoints for vancomycin are:
1. susceptible, ≤4 mg/L;
2. intermediate, 8-16 mg/L;
3. resistant, ≥32 mg/L.[4]

In 2002, vancomycin-resistant *S. aureus* (VRSA) (MIC ≥32 mg/L) were recovered from two patients in the USA. Unlike GISA and hGISA, these isolates carried the *vanA* gene (*vanA* encodes vancomycin resistance in enterococci).[5] To date, these are the only VRSA reported but GISA have been reported from Japan, USA, Korea, South Africa, South America and Europe.[2] GISA isolates are still rare but hGISA isolates are reported more widely although their clinical relevance is unclear.[1] Some reports associate hGISA with clinical failure, others suggest that they are over-reported but their real importance may be that they represent a pre-GISA state which could, in time, lead to treatment failure.[2, 6]

MRSA recovered from blood cultures taken in Irish hospitals that participate in the European Antimicrobial Resistance Surveillance System (EARSS) are sent to the National MRSA Reference Laboratory (NMRSARL) for extended investigation of antimicrobial resistance. NMRSARL screens all EARSS MRSA isolates for glycopeptide resistance with

- the E-test (AB BioDisk) macro-method using both vancomycin and teicoplanin
- by agar screening on brain heart infusion agar containing 6 mg/L vancomycin (BHIV6).[1, 3]

Any isolate exhibiting an E-test macro-method value of ≥6 mg/L or capable of growth on BHIV6 is further investigated by E-test MIC determination on Mueller-Hinton agar and, if necessary, by broth microdilution MIC using the NCCLS method to out-rule GISA[4]. Isolates exhibiting E-test macro-method values of 8 mg/L for both vancomycin and teicoplanin or 12 mg/L for teicoplanin alone are possible hGISA.[7] Between January 2002 and May 2003, eight possible hGISA were detected among 553 isolates tested. These isolates were sent for population analysis profile/area under the curve (PAP-AUC) ratio determination to the Bristol Centre for Antimicrobial Research and Evaluation (BCARE).[3] BCARE's criterion for hGISA is a PAP-AUC ratio of 0.9 - 1.3.[3]

Two of these isolates yielded PAP-AUC ratios of 0.92 and 0.94, respectively. This is the first confirmed report of hGISA in Ireland. These hGISA isolates were recovered in different hospitals; one exhibited the antibiogram-resistogram (AR) type, AR13.1 and was fusidic acid resistant; the other was a variant of either of two AR types, AR13 or AR14. Pulsed field gel electrophoresis (PFGE) patterns obtained following digestion of chromosomal DNA with the restriction endonuclease *Sma*I showed <60% similarity between the isolates suggesting that they are not closely related. In one of the hospitals, a subsequent suspect hGISA isolate was recovered from a different patient two months after the initial isolate. Both isolates in this hospital exhibited indistinguishable AR patterns and closely related PFGE profiles. Information on the patients' clinical outcome is being sought and further investigation is underway at NMRSARL to confirm other possible hGISA isolates from earlier EARSS MRSA and from previous studies of MRSA in Ireland.[8, 9]

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Acknowledgements

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Rubella Notifications

The NVRL have reported 2 samples positive for rubella IgM in the week ending October 24th. To date in 2003, 54 cases of rubella have been notified to NDSC which is double that reported during the same period in 2002. It may represent an increase in sampling of rash illness due to the increased measles activity seen this year or may be the start of an increase in rubella. We would be interested to hear if anyone becomes aware of an increase in laboratory-confirmed cases of rubella.

Salmonella Monthly Report (September 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, NSRL.

Health Board	E	M	MW	NE	NW	SE	S	W	Total
S. Agona	0	0	0	0	0	0	0	1	**1**
S. Bovismorbifican	0	0	0	1	0	0	0	0	**1**
S. Braenderup	0	1	0	0	0	0	0	0	**1**
S. Bredeney	1	0	0	0	0	0	0	0	**1**
S. Enteritidis	18	3	3	4	1	6	6	2	**43**
S. Hadar	0	0	1	6	0	0	0	0	**7**
S. Havana	0	0	1	0	0	0	0	0	**1**
S. Kentucky	1	0	0	0	0	0	0	0	**1**
S. Kottbus	0	1	0	0	0	0	0	0	**1**
S. Newport	1	1	0	0	0	0	0	0	**2**
Paratyphi A	0	0	0	0	0	0	1	0	**1**
S. Stanley	1	0	0	0	0	0	0	0	**1**
S. Tennessee	0	0	0	0	0	1	0	0	**1**
S. Typhimurium	9	3	1	0	0	4	2	7	**26**
S. Virchow	1	0	1	0	0	1	0	0	**3**
Total	32	9	7	11	1	12	9	10	**91**