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Review on Influenza Virus

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ABSTRACT

Emerging infections account for at least 12% of all human pathogens. From that influence is one of the pandemic disease that causes economic destruction and causes high morbidity mainly in human as well in birds and swine. Three types of influenza viruses affect people, called Type A, Type B, and Type C. Usually, the virus is spread through the air from coughs or sneezes. This is believed to occur mostly over relatively short distances. Influenza has ability to Antigenic Changes through Antigenic Drift and Antigenic Shift. Due to the high mutation rate of the virus, a particular influenza vaccine usually confers protection for no more than a few years. So, that prevention and control before occurrence outbreak is very important.

Keywords: Emerging infections, influenza, virus and human pathogens.

INTRODUCTION

An emerging infectious disease (EID) is an infectious disease whose incidence has increased in the past 20 years and could increase in the near future. Emerging infections account for at least 12% of all human pathogens [1]. EIDs are caused by newly identified species or strains (Severe acute respiratory syndrome, HIV/AIDS) [2] that may have evolved from a known infection (influenza) or spread to a new population (West Nile fever) or to an area undergoing ecologic transformation (Lyme disease). Influenza is considered to be one of the life threatening infectious diseases. In some countries seasonal influenza affects annually up to 40% of the population and 500 million people die from it worldwide every year [2]. Influenza viruses belong to the *Orthomyxoviridae* family. Flu symptoms include fever, dry cough, sore throat, headache, extreme tiredness and body aches. These symptoms usually begin suddenly and might be severe enough to stop your daily activities. The diagnosis of influenza is usually suspected on the basis of characteristic clinical findings, particularly if influenza has been reported in the community. It is extremely important to have early detection and warning systems and prevention measures in place as part of an effective strategy for all infections.

ETIOLOGY

Influenza is a single-stranded, helically shaped, RNA virus of the orthomyxovirus family. Basic antigen types A, B, and C are determined by the nuclear material. These viruses are only distantly related to the human parainfluenza viruses, which are RNA viruses belonging to the paramyxovirus family that are a common cause of respiratory infections in children such as croup [3] but can also cause a disease similar to influenza in [4]. The specific strains of influenza change frequently, necessitating parallel changes in the seasonal influenza vaccine. Since 1977, three types of influenza viruses had been in circulation in humans: influenza A(H3N2), influenza A(H1N1), and influenza B.

Type	Subtype	Prototype
A	H1N1	A/PR/8/34
		A/NJ/8/76
	H2N2	A/JP/305/57
B	H3N2	A/HK/1/68
	None	B/Lee/40
C	None	C/Taylor/47

EPIDEMIOLOGY

Avian species, specifically shorebirds and waterfowl, are a reservoir of influenza

A virus of different subtypes in nature. With a few notable exceptions, influenza infections in these hosts are asymptomatic and are limited to the gastrointestinal and/or the respiratory tract [5]. Influenza viruses infecting these hosts appear to be in evolutionary stasis compared with those infecting humans. High titers of influenza viruses are excreted from the gastrointestinal tract of infected birds, and viruses excreted into bodies of water can survive for several weeks. There is a recurring supply of susceptible birds each season

and up to 30% of juvenile birds are infected [6]. The viruses spread to other susceptible avian species, such as turkeys, presumably through contamination of water in the farms along the flyways of migratory bird populations [7] [8]. Viruses that are a pathogenic for shorebirds or waterfowl can be pathogenic for certain other avian species. For example, influenza A H5N1 viruses isolated from humans and chickens in Hong Kong in 1997 were highly pathogenic for chickens, and although they replicated to low titer in experimentally infected ducks, they did not cause disease signs in these birds. The viruses isolated from ducks in the market were lethal for experimentally inoculated chickens. Alternatively, a previously virulent influenza virus can acquire specific sequence changes, such as losing potential glycosylation sites or gaining multiple basic amino acid sequences in the connecting peptide of the HA, that confer virulence[9].

The influenza season is largely finished in the temperate countries of the northern hemisphere and most countries in the northern temperate zone have stopped weekly reporting or moved over to out of season surveillance schedules. In the tropical zone, the countries to report notable influenza activity are Bolivia, Brazil, and Honduras in the Americas; Ghana in sub-Saharan Africa; southern China, including Hong Kong Special Administrative Region, and Viet Nam in Asia. The influenza season has commenced in most temperate countries of the southern hemisphere. In Argentina, however, influenza remains nearly undetectable. Influenza A(H3N2) viruses were the most commonly reported type/sub-type in recent weeks in the Southern Hemisphere temperate region in Chile, South Africa, and Australia; however, significant numbers of influenza type B were also reported in South Africa and to a lesser extent, Australia. Very few influenza A(H1N1)pdm09 viruses have been reported with exception of Paraguay and some countries in Central and tropical South America[10].

The incidence of seasonal influenza typically increases in the late autumn and begins to decline in mid spring. In the Northern hemisphere, this corresponds to November through March; in the Southern Hemisphere, this corresponds to April through September. In tropical countries, influenza occurs sporadically throughout the year, but more so in the rainy periods. Localized outbreaks of seasonal influenza also occur in inter-pandemic years, particularly when strains of virus penetrate communities with little or no preexisting immunity to the circulating virus. The reason for seasonality remains unclear. Because the primary mode of transmission is by large droplet aerosols, increased crowding in the colder months, the return to schools and university dormitories and the start of military recruit courses have been suggested as contributing factors. Fomites may serve as a secondary mode of transmission, and it has also been suggested that the higher intensity of sterilizing ultraviolet light in the summer months may serve to reduce the environmental burden of virus. Dry environments, such as those that prevail during the winter months, are also known to increase transmission for unknown [11].

RISK FACTOR

All of the genes found in the H5N1 viral strain in Hong Kong originated from avian viruses.^{1, 2} While H5N1 has not yet demonstrated the ability to transmit efficiently from person to person, the high case fatality associated with reported infection, ongoing spread of the virus in bird populations, and the potential for influenza viruses to mutate and adapt to other hosts mean H5N1 remains a continuing public health concern. As of August 10, 2012, H5N1 infection had been detected in 608 individuals in 15 countries.³

The number of human cases is not evenly distributed throughout the world, and the age/gender distribution varies by country. The largest numbers of human cases reported have been from Indonesia, Vietnam, and Egypt, each having reported more than 100 cases. No human cases have yet been reported in Western Europe or the Americas. Although the apparent case fatality rate (CFR) of H5N1 is high (approximately 59%), this may be an overestimate of the true CFR because any milder cases may never be identified under current surveillance systems in countries affected by H5N1. To date, H5N1 remains an avian epidemic with rare and sporadic spillover into the human population and other species. The predominant modes of transmission from poultry to humans remain incompletely understood, and limited information on how infected individuals were exposed has restricted our ability to evaluate risk factors for human infection and implement more refined risk reduction measures [9]. We used the age categories that were most commonly reported in the original articles: >65 years for elderly, <18 years for children, 2 to <5 years, <2 years, and <6 months. If other categories were reported, we chose the closest to these cut-offs. We compared other ethnic groups with white participants. Definitions of comorbidities by the original studies were used. Obesity was defined as a body mass index of >30 kg/m² or as defined by the original studies. Factors associated with death or hospitalization Of those admitted to intensive care, up to 98% of patients and 91% of fatalities of pdm (H1N1) 09 associated deaths were related to at least one co-morbidity [6,7,9-11] such as; age, chronic lung disease, obesity, pregnancy, diabetes, cardiac disease and immunosuppression. However a study of those admitted to ICUs in Australia and New Zealand found that 31.7% had no underlying condition .Age: Generally patients with a non-severe clinical outcome were younger than those admitted to ICUs, who were also younger than those who died. According to one Canadian study, the median age was 23 years for all patients, 18 years for those with non-severe outcome, 34 years for those admitted to ICUs and those aged over 45 years were significantly more likely to die . The greatest frequency of hospital admissions was in children aged 0-1 year followed by adults in the 50-64 year age group. While studies suggest that children and young people had the highest cases of hospital admission, CFRs were highest in the over 65 year age group .Lung disease: Chronic lung disease, asthma in particular, appears to be a common co-morbidity in severe cases of pdm(H1N1)09 infection including those requiring intensive care and those resulting in death. Data on admissions to ICUs related to H1N1 influenza infection in Australia and New Zealand in 2009 showed that 32.7% of admissions had asthma or chronic pulmonary disease and in Canada 41.1-47% of the critically ill also displayed these co-morbidities. In Canada it was also shown that 22.6-38% of the critically ill were or had been smokers Smith [11]. In California in 2009, 41% of fatal influenza cases had chronic lung disease while in Germany it was reported to be 23.1% [15]. Obesity: Obesity has commonly been reported in ICU submissions and patients dying from pdm (H1N1)09 and has been identified as a predictor of outcome in acute respiratory infection [19], possibly as a consequence of a defective cellular immune response to infection . Of those admitted to an ICU in Australia and New Zealand in 2009, 28.6% (of which data was available) had a BMI of 35 kg/m² or greater. In Canada 33.3% of the critically ill were obese (BMI ≥30), and in one region of Canada the figure was as high as 62%. In California, 66% of fatal adult cases involved obesity (BMI≥30) while in Germany the 23.1% of reported fatalities involved obesity (BMI >30).

Pregnancy: Pregnancy has frequently been reported in cases of hospital admission related to pdm (H1N1) 09 into ICUs. Pregnancy conveys a 4-5 fold increased rate of serious illness and

hospitalisation with influenza. During previous influenza outbreaks, pregnancy has also been associated with increased mortality and morbidity; particularly if infection occurs during the third trimester

TRANSMISSION

Several factors can contribute to the spread of AI viruses including globalization and international trade (legally and illegally), marketing practices (live bird markets), farming practices and the presence of the viruses in wild birds. Wild birds normally can carry avian influenza viruses in their respiratory or intestinal tracts and usually do not get sick. Wild birds have historically been known as reservoirs for AI viruses, mostly of low pathogenicity. Around the world, surveillance measures have been put in place to monitor occurrence and characteristics of AI viruses in wild birds. In wild birds, it is common during routine testing to find some influenza viruses. The vast majority of these viruses do not cause disease. In studying current HPAI H5N1 outbreaks, the exact role of wild birds in spreading the virus over long distances is still not fully understood in all situations. Generally, there are many uncertainties about the wild species involved, the migratory routes used and, above all, the possibility that some species could become permanent reservoirs of the H5N1 virus, with carriers showing no

CLINICAL SIGNS OF THE DISEASE.

AI viruses can be spread through direct contact with secretions from infected birds, especially faeces or through contaminated feed, water, equipment and clothing. Apart from being highly contagious among poultry, avian influenza viruses are readily. The incubation period for influenza is usually 2 days, but can vary from 1 to 4 days. Influenza illness can vary from asymptomatic infection to severe. In general, only about 50% of infected persons will develop the classic clinical "Classic" influenza disease is characterized by the abrupt onset of fever, myalgia, sore throat, nonproductive cough, and headache. The fever is usually 101°-102°F, and accompanied by prostration (bedridden). The onset of fever is often so abrupt that the exact hour is recalled by the patient. Myalgias mainly affect the back muscles. Cough is believed to be a result of tracheal epithelial destruction. Additional symptoms may include rhinorrhea (runny nose), headache, substernal chest burning and ocular symptoms (e.g., eye pain and sensitivity to light). Systemic symptoms and fever usually last from 2 to 3 days, rarely more than 5 days. They may be decreased by such medications as aspirin or acetaminophen. Aspirin should not be used for infants, children, or teenagers because they may be at risk for contracting Reye syndrome following an influenza infection. Recovery is usually rapid, but some patients may have lingering asthenia (lack of strength or energy) for several weeks.

DIAGNOSIS

Respiratory virus testing should be considered in individuals presenting during influenza season with fever and influenza-like symptoms of myalgia, arthralgia, headache, and/or sore throat. Influenza testing is not needed for all patients with signs and symptoms of influenza to make antiviral treatment decisions. A clinical diagnosis of influenza can be made for outpatients with signs and symptoms consistent with influenza. For outpatients and emergency room patients, results of diagnostic testing are not available in a timely manner to inform clinical decision making. Testing is appropriate for hospitalized inpatients, especially if a positive test would result in a change in clinical management.

Nasopharyngeal swab specimens are the preferred specimen for the purpose of respiratory virus testing. A new influenza A/B/RSV PCR (#7255) is the diagnostic test of clinical diagnosis of influenza. Although the clinical presentation of influenza is similar to illness caused by other respiratory pathogens, when influenza is circulating in the community, several studies show that the presence of cough and high fever of acute onset is likely to be associated with influenza infection [22-25]. In a retrospective study of 3744 unvaccinated adults and adolescents with influenza-like symptoms, who were enrolled in a clinical trial of an anti-influenza drug, patients presenting with cough and fever (37.8°C) were likely to have laboratory confirmed influenza (positive predictive value (PPV), 79%) [22]. The probability of a patient having confirmed influenza increased further with increasing fever and if patients presented acutely (within 36 to 48 hours of onset). Similarly, in an additional study of general practitioners in three different clinics, the PPV of the presence of fever (38°C) and cough for laboratory confirmed influenza was 86.8%. However, the clinical diagnosis of influenza was imperfect (negative predictive value (NPV)). Factors that may potentially confound the clinical diagnosis include: the circulating virus strain, preceding use of antipyretics; duration of symptoms at presentation, and the patient's age, vaccination status and any underlying illnesses. It is important to stress that these studies were conducted during periods when laboratory confirmed influenza cases were documented in the community. In the absence of a known influenza epidemic, the PPV of the same clinical criteria was only 44% [25].

This highlights the importance of the season in which the patient presents and of notification of health care providers of the results of active influenza surveillance in the community. Indeed, during an influenza epidemic the intuition of general practitioners for the presence of influenza infection is one of the most accurate diagnostic tools. Specifically, when influenza is known to have arrived at UCLA. Laboratory diagnosis of influenza. Given the potential accuracy of clinical diagnosis of influenza during influenza season, one might question the role of the laboratory in influenza diagnosis. In contrast to other 'flu-like illnesses', influenza is associated with higher morbidity and mortality, is potentially preventable by vaccination and chemoprophylaxis and is treatable by specific antivirals. In addition, it is important for both the patient's and public health perspective to differentiate influenza from 'flu-like illness' caused by other respiratory pathogens. Laboratory isolation of influenza provides culture confirmation of the arrival of influenza in a community.

This is valuable information for the physician as it greatly enhances the accuracy of their clinical diagnosis. Physicians in close communication with the microbiology laboratory and aware of the arrival of influenza season should be able to diagnose most influenza infections clinically. This should reduce unnecessary additional laboratory testing and inappropriate antimicrobial treatment [7]

Serological tests: As all influenza A viruses have antigenically similar nucleoprotein and matrix antigens, these are preferred targets of influenza A group serological methods. Agar gel immunodiffusion tests can be used to detect antibodies to these antigens. Concentrated virus preparations containing either or both type of antigens are used in such tests. Not all species of birds develop demonstrable precipitating antibodies. Enzyme-linked immunosorbent assays have been used to detect antibodies to influenza A type-specific antigens in either species-dependent (indirect) or species-independent (competitive) test formats. Haemagglutination inhibition tests have also been employed in routine diagnostic

serology, but it is possible that this technique may miss some particular infections because the haemagglutinin is subtype specific.

CONTROL

It is extremely important to have early detection and warning systems and prevention measures in place as part of an effective strategy for AI. This needs to be coupled with similar efforts placed on preparing for a potential outbreak. round the world, surveillance measures have been put in place to detect the presence of infection in poultry according to OIE standards for the surveillance of avian influenza (OIE Terrestrial Animal Health Code). Additionally, surveillance programmes monitor the occurrence, prevalence and characterisation of AI viruses found in wildbirds. Wild bird surveillance considers different migratory flyways and particularly at mingling points for migrating birds from different continents. It is essential for poultry producers to maintain biosecurity practices to prevent introduction of the virus in their flock. In the United States, the primary option for reducing the effect of influenza is immunoprophylaxis with vaccine. Inactivated (killed virus) influenza vaccine and live, attenuated influenza vaccine are available for use in the United States (see Recommendations for Using Inactivated and Live, Attenuated Influenza Vaccine). Vaccinating persons at high risk for complications and their contacts each year before seasonal increases in influenza virus circulation is the most effective means of reducing the effect of influenza [11]. Vaccinating residents and healthcare personnel (HCP) is the only truly effective strategy for influenza control. Although vaccination may not be 100% preventative, studies have shown that an affective vaccination program reduces influenza-related complications and deaths, and can also lower HCP absenteeism.

An influenza prevention program should include the following: Annual review of the CDC and CDPH influenza vaccination recommendations (<http://www.cdc.gov/flu/professionals/vaccination/>). Well-defined influenza policies and procedures to be reviewed with the infection prevention committee prior to the influenza season. A designated "Influenza Vaccination Week." Choose one week where influenza vaccination is featured to vaccinate as many residents and HCP as possible. The CDC currently recommends that vaccination programs begin as soon as flu vaccine is available at the facility, even as early as August, and continue through the end of April of the following year. Education for all residents, visitors, and HCP concerning the importance of vaccination, recognizing symptoms of infection, facility policies regarding work restrictions when ill, visitor restrictions, appropriate respiratory precautions, and hygiene cough etiquette. A campaign to encourage employees to vaccinate their family members. A campaign to encourage visitors to be vaccinated. Healthcare Personnel Vaccination Develop and distribute written information that describes the benefits of influenza vaccination and the possible side effects of the vaccine. Strongly encourage influenza vaccination for all HCP including administrative personnel. Vaccinate new employees or request proof of vaccination prior to their start date. Resident Vaccination Vaccinate current residents during the designated seasonal influenza vaccination week. Vaccinate new admissions at any time between August and April, if not already vaccinated. Consider having residents or the resident's advocate (e.g., a family member, friend, or ombudsman) sign a "Consent to be Vaccinated" form. Assess immunization history and ensure that updated pneumococcal vaccination standing orders are in place for residents ≥ 65 years [8].

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