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H1N1 influenza is here

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Summary
On 11 June WHO announced that the spread of H1N1 had reached pandemic phase 6. Since then countries around the world have increased their planning and preparedness for the continuing pandemic. Guidelines have been issued and circulated but confusion continues over their interpretation. We need to remind ourselves of the principles behind control measures and educate and reinforce standard infection control procedures in the acute hospital setting.

LEADER

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Since the first case of novel influenza infection was announced in Mexico in April 2009, swH1N1 Influenza A — a mosaic of swine/bird/human Influenza A1 — has spread across the world at lightning speed. The outbreak of acute (though mild) respiratory illness originally emerged in the La Gloria region of Veracruz in Mexico affecting 28% of 2155 people. By the end of June 2009, the strain had already spread to more than 120 countries and more than 70,000 cases of infection had been reported. The USA has reported the majority of cases, with more than 27,000 confirmed. This is likely to be the tip of the iceberg since surveillance systems are not widespread in any country.

The World Health Organization (WHO) quickly responded to the pandemic threat, announcing a move from phase 4 to 5 of the pandemic phases in early June. This was followed by final verification of phase 6 status on 11 June, with continued identification of new cases in many countries and sustained human-to-human transmission of swH1N1. This is the first influenza pandemic since summer 1968 when H3N2 spread across Southern Asia to Hong Kong and beyond causing 38,000 deaths. The clarion call from WHO set in motion a series of actions by every government and healthcare system across the world, requiring them to plan and prepare for the worst.

There has been a continued flurry of documents and communications emerging at a speed almost equaling that of the spread of the virus. Having had two recent ‘near misses’, first with the emergence of severe acute respiratory syndrome caused by a novel coronavirus (SARS-CoV) in 2002–3 and then the ongoing spread of H5N1 since 1999 (particularly in the Far East), many of us already had guidance prepared. Therein lies a problem,
in that swH1N1 is neither SARSCoV nor H5N1. The natural history of these viruses is quite different. It is unlikely that patients with SARS were infectious before they became ill, which is not the case with swH1N1 (and every other influenza virus causing illness in humans). H5N1, on the other hand, does not easily spread from person-to-person, possibly due to the cell receptors it selects to initiate infection.

From the acute hospital perspective, the major challenges include prevention of nosocomial spread to other patients and staff in healthcare facilities. It is important to protect healthcare staff so that they avoid infection and can continue to perform their jobs. Other issues include the use of isolation facilities, clear guidance on cohorting and prudent and appropriate antibiotic prescribing for those patients found to have post-influenza bacterial pneumonia.

Guidelines are essential but some documents now circulating have 70–100 pages to sift through. Even the nomenclature of the virus varies between different publications and causes confusion in the hospital, let alone among the general public. There are some basic scientific facts about influenza virus which will help us. Unlike guidelines, these are not subject to the same difficulties with interpretation. We know a great deal about influenza viruses, since there have now been three previous pandemics in the last hundred years, the last during 1968. The experience gained from these is, perhaps, a more useful context from which to view the current pandemic, rather than compare with the 1918 pandemic when the world was a different place. Right now, swH1N1 is behaving in a similar way to pandemics, rather than compare with the 1918 pandemic, when the world was a different place in that swH1N1 is neither SARSCoV nor H5N1. The natural history of these viruses is quite different.

We know influenza viruses are enveloped viruses. They are readily inactivated by physical and chemical agents. Assuming viral shedding follows the usual pattern for such viruses, surfaces should be easily decontaminated. Infectious droplets generated in aerosols and landing on environmental surfaces will be inactivated by simple detergent treatment. Thus, standard infection control decontamination procedures are entirely appropriate in the current situation, but once again, confusion has arisen because guidelines are not ‘standard’ or consistent. It is, for example, surprising to notice that WHO recommend decontamination with disinfectant, specifically, chlorine at strength of 500 ppm and not detergent. Indeed, usage of disinfectants during this pandemic has been advised in other European countries and in the USA.

Inevitably, the first report of swH1N1 infection among healthcare workers (HCWs) has already been published. Of 13 HCWs deemed to have acquired infection in the healthcare setting, only three had consistently used PPE. There is another strand to our strategy to prevent infection—antiviral therapy. Two neuraminidase inhibitors are licensed: oseltamivir and zanamivir. There is modest evidence that oseltamivir (Tamiflu™), if given orally early enough is effective for prophylaxis. Zanamivir is administered by inhalation and can be used as a back-up. Both drugs work by interfering with release of virus from infected cells. Governments for some time have been establishing a stockpile of these drugs for post-exposure prophylaxis in the community and hospital. Much reliance is being put on oseltamivir for prophylaxis and treatment of the pandemic strain, but no clear or consistent guidance is available defining ‘key workers’ in an institution/country qualifying for antiviral prophylaxis. This continues to generate considerable debate with significant political overtones. From the infection control perspective, antiviral resistance to the drugs can be
predicted to emerge, especially if uncontrolled distribution occurs.

Immunisation of HCWs is also likely to be effective provided we can achieve a reasonable uptake, a target that has always been incredibly difficult to achieve. Despite vaccine effectiveness, HCWs historically under-use it. Uptakes are poor (from 2 to 32%) and attitudes will have to be changed if vaccine strategies are to be effective. Seasonal influenza vaccines include human influenza viruses of subtypes H1N1 and H3N2, and an influenza B virus. The composition of these vaccines is revised every year, in order to accommodate any molecular changes in circulating strains. The vaccines contain influenza virus, which is grown in eggs or cell culture, then inactivated so that it cannot cause infection but will induce protective antibodies. New approaches are being used to make vaccines and good progress is being made with the production of a vaccine against the new strain. Governments have already commissioned stocks of new vaccine. When safety trials have been carried out a vaccine could be available in the autumn. This will be too late, however, for Southern hemisphere countries, now experiencing their influenza season.

In summary, we need to continue to educate our HCWs, with simple messages aimed at clarifying and reinforcing current infection control recommendations. We need a ‘gold standard’ for policies—do we follow guidance from WHO or from national bodies such as the relevant Health Protection Agency in the UK? Should guidance be endorsed by UK governments? Such policies need to be locally accessible, comprehensive and acted upon. Can we develop a rapid response system to keep our policies up-to-date and effective against a virus that spreads and changes at remarkable speed? To date, we have seen few deaths due to swH1N1 but although this virus is causing milder illness compared with its predecessors there is no room for complacency. Early evidence from Chile suggests that swH1N1 is replacing the circulating seasonal strains. Reports of swH1N1 resistant to oseltamivir are emerging (at the time of writing), which serves as a reminder of previous experience with resistant H1N1 during 2008–9. Reassortment of viral components during seasonal influenza circulation is a real possibility, with a shift towards increasing pathogenicity. Influenza pandemics are fickle. The virus evolves, patterns vary from country to country and cases come in waves. We do, however, have the knowledge, experience and tools we need to prevent this virus spreading in the hospital setting if we apply them. It is back to basics.

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