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Influenza A (H1N1 09)

Public health lessons and questions

Influenza A (H1N1 09) has been in Australia for 2 months and, in a world that had been preparing for a potentially dramatic outbreak due to H5 avian influenza, it has challenged global and national planning assumptions, definitions of pandemic influenza and our public health interventions. It has also highlighted how much we have yet to learn about both pandemic and seasonal influenza.

H1N1 has spread faster than any previous virus, with a degree of international spread in 6 weeks that has taken other pandemic viruses 6 months to achieve.[1] Interestingly, the current virus does not yet have all the genetic features that maximise human transmissibility, and in particular the phenotype of the polymerase basic protein 2, which has been present in all three previous 20th century pandemic viruses.[2] Rapid spread in spite of this, highlights the importance of other factors, including the massive scale of both international and national travel that characterises 21st century life. The concept and implementation of border control as a public health measure to contain influenza has previously been questioned by modelling studies[3] and this is reinforced by recent experience.

Once in Australia, H1N1 quickly caused outbreaks among school children. This phase tested the public health approach to school closure, the isolation of cases and contacts, and the use of antiviral drugs. General practitioners were immediately at the forefront of the response and concerns have been raised about possible deficiencies and delays in the implementation of the Australian Health Management Plan for Pandemic Influenza (AHMPPI).[4,5] Health authorities quickly established various methods to communicate with GPs, however the effectiveness of the large number of bulletins, emails and websites – at a time of rapid change and uncertainty – requires evaluation.

Another major challenge during the early phase was the delivery of a complicated public health message to the general population. The virus was clearly causing explosive outbreaks and the risk of rapid spread was high. Although, in most cases, the illness was mild and short lived, there was a substantial group of individuals in the community who, if they became infected, were at high risk of developing significant complications.

Just as claims of a beat up about the severity of the pandemic were being made, the first cases of severe illness and deaths were reported. This phase of severe illness has raised questions about the pathogenicity of H1N1 and the mechanisms by which it causes complications. Are the numbers of severe cases a reflection of the increased total number of people who have been infected, or is the pandemic virus more pathogenic than seasonal flu? The answer is not known. Public debate about whether or not this pandemic virus is behaving differently from seasonal influenza, 'It's just another flu virus except we don't have immunity', also underscores the gaps in our knowledge about seasonal flu: its method of spread in different groups of people and how to limit this; the morbidity and mortality in previously well and at risk groups and how this occurs; the efficacy of vaccination; and how to best use antiviral drugs.

The early arrival of H1N1 in Australia at the beginning of our influenza season has emphasised the shortcomings of current vaccine production, in particular the long lead times. Oseltamivir resistance has been reported in pandemic H1N1 overseas, although at a frequency that suggests it is a sporadic event and without any clinical consequences.[1] Nonetheless the spectre of wide spread resistance to antiviral drugs is another challenge for our public health response to pandemic influenza.

We are visited by influenza every year, but still don't know our unwelcome guest very well. It is imperative that the experiences of this pandemic, particularly of those involved in community and hospital management, are collected, carefully analysed and acted on.

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