

(H1N1) became resistant to both oseltamivir and amantadine in a short span of 1 month. Oseltamivir-resistant A/Brisbane/59/2007-like clade 2B virus that had reassorted with A/Hong Kong/2652/2006-like clade 2C virus had apparently spread in the community and to other regions of the world. The possibility of reassortment with pandemic (H1N1) 2009 virus is a major concern. Resistance to oseltamivir of pandemic (H1N1) 2009 virus will compromise its use in treatment and render the billion-dose stockpile useless. Although the recently detected oseltamivir-resistant pandemic (H1N1) 2009 virus in Hong Kong was not a reassortant virus (4,5), we will continue to closely monitor antiviral drug resistance among circulating viruses, including pandemic (H1N1) 2009 virus and seasonal influenza virus A (H1N1), as well as influenza A (H3N2) viruses, to track how antiviral resistance evolves.

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#### References

- Cheng PKC, Leung TWC, Ho ECM, Leung PCK, Ng AYY, Lai MYY, et al. Oseltamivir- and amantadine-resistant influenza viruses A (H1N1). *Emerg Infect Dis.* 2009;15:966–8. DOI: 10.3201/eid1506.081357
- Hurt AC, Ernest J, Deng Y, Iannello P, Besselaar T, Birch C, et al. Emergence and spread of oseltamivir-resistant A(H1N1) influenza viruses in Oceania, South East Asia and South Africa. *Antiviral Res.* 2009;83:90–3. DOI: 10.1016/j.antiviral.2009.03.003
- Centre for Health Protection, Department of Health, The Government of the Hong Kong Special Administrative Region. Monthly summary tables of influenza virus isolation 2009 [cited 2009 Jul 31]. Available from [http://www.chp.gov.hk/data.asp?lang=en&cat=5&dns\\_sumID=377&id=292&pid=44&ppid=26](http://www.chp.gov.hk/data.asp?lang=en&cat=5&dns_sumID=377&id=292&pid=44&ppid=26)
- World Health Organization. Global Alert and Response (GAR). Viruses resistant to oseltamivir (Tamiflu) identified [cited 2009 Jul 31]. Available from [http://www.who.int/csr/disease/swineflu/notes/h1n1\\_antiviral\\_resistance\\_20090708/en/index.html](http://www.who.int/csr/disease/swineflu/notes/h1n1_antiviral_resistance_20090708/en/index.html)
- Leung TWC, Tai ALS, Cheng PKC, Kong MSY, Lim W. Detection of an oseltamivir-resistant pandemic influenza A/H1N1 virus in Hong Kong. *J Clin Virol.* 2009;46:298–9. DOI: 10.1016/j.jcv.2009.08.004

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## Pandemic (H1N1) 2009 Reinfection, Chile

**To the Editor:** Since March 2009, influenza A pandemic (H1N1) 2009 has spread worldwide (1), and in South America, Chile was 1 of the countries most affected by the pandemic, with 21.4 cases among every 1,000 persons. Treatment guidelines in Chile recommended antiviral drug treatment with oseltamivir or zanamivir for 5 days for all patients with confirmed or suspected virus subtype H1N1 infection (2). In persons with seasonal influenza, specific antibody responses reach peak titers by 4 weeks after infection and confer protection against the infecting strain and closely related strains (3). Reinfection is rarely seen in nonpandemic influenza A. We report on 3 patients with confirmed influenza A pandemic (H1N1) 2009 reinfection after successful treatment with oseltamivir.

Patient 1, a healthy 14-year-old girl, had fever, sore throat, and nasal

congestion on clinical examination. Pandemic (H1N1) 2009 infection was diagnosed by viral culture and confirmed by PCR specific for subtype H1N1 (LightMix Kit Inf A swine H1; TIB MOLBIOL, Berlin, Germany, for Roche Diagnostic, Indianapolis, IN, Light Cyclor 2.0 instrument). The patient received oseltamivir, and symptoms resolved 48 hours after treatment. Twenty days later, fever, muscle aches, and vomiting developed in the patient. Influenza A virus was isolated by viral culture. The patient received a preliminary diagnosis of seasonal influenza and was treated with amantadine. She recovered from the infection before PCR results confirmed it was caused by pandemic (H1N1) 2009 virus.

Patient 2, a 62-year-old woman, experienced a high fever, cough, and nasal congestion during a prolonged hospitalization for bowel resection after intestinal ischemia. Pandemic (H1N1) 2009 was confirmed by PCR and viral culture. Oseltamivir was administered 5 days after the onset of symptoms, and the symptoms resolved within the following 5 days. The patient had a new episode of fever, productive cough, and bronchial obstruction 2 weeks later while still hospitalized. Culture results were positive for influenza, and PCR results were positive for pandemic (H1N1) 2009. The patient was again treated with oseltamivir, and PCR results were negative for influenza after 48 hours of antiviral treatment.

Patient 3, a previously healthy 38-year-old man, underwent mitral and aortic valve replacement while hospitalized for acute mitral and aortic endocarditis due to *Staphylococcus aureus*. Eleven days after surgery, he had a sore throat, nasal congestion, cough, and low-grade fever. PCR test results were positive for pandemic (H1N1) 2009. The patient received oseltamivir, and respiratory symptoms resolved within 5 days. He was discharged from the hospital but was

readmitted 18 days later with nasal congestion, cough, and high fever. PCR results were again positive for pandemic (H1N1) 2009, and the patient was successfully treated with oseltamivir.

Patient 2 and probably patient 3 acquired their infections while hospitalized, suggesting potential nosocomial transmission. No other respiratory viruses were found in any of these patients. The viral isolates were all tested (LightMix for detection of influenza A virus oseltamivir resistance [H274Y]; TIB MOLBIOL) for possible resistance to oseltamivir, but none had the resistance-implicated H274Y mutation in the neuraminidase gene.

Shedding of seasonal influenza A virus ceases within 5–7 days during natural infection and during infections treated with neuraminidase inhibitors (4). Although clearing of virus after the first infection was not documented in the 3 patients described here, it is unlikely that virus persisted between the 2 episodes of influenza since each of the patients fully recovered after specific antiviral drug treatment. However, we cannot rule out that patient 2 may have never cleared the virus due to her immune suppression.

As described by mathematical modeling (5), the 3 patients described were susceptible to reinfection with pandemic (H1N1) 2009 due to the high rate of community infection and to their incomplete immunologic protection within the period of reexposure. During the current pandemic of influenza subtype H1N1, healthcare workers and patients should be aware that symptomatic reinfection might occur after a first episode of infection.

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## References

1. World Health Organization. Pandemic (H1N1) 2009—update 58 [cited 2009 Jul 12]. Available from [http://www.who.int/csr/don/2009\\_07\\_06/en/index.html](http://www.who.int/csr/don/2009_07_06/en/index.html)
2. Ministerio de Salud. Influenza [in Spanish] [cited 2009 Sep 14]. Available from [http://www.redsalud.gov.cl/portal/url/page/minsalcl/g\\_varios/influenza.html](http://www.redsalud.gov.cl/portal/url/page/minsalcl/g_varios/influenza.html)
3. Treanor JJ. Influenza virus. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases. 6th ed. New York: Elsevier; 2005. p. 2060–85.
4. Aoki FY, Boivin G. Influenza virus shedding: excretion patterns and effects of antiviral treatment. *J Clin Virol*. 2009;44:255–61. DOI: 10.1016/j.jcv.2009.01.010
5. Gökaydin D, Oliveira-Martins JB, Gordo I, Gomes MG. The reinfection threshold regulates pathogen diversity: the case of influenza. *J R Soc Interface*. 2007;4:137–42. DOI: 10.1098/rsif.2006.0159

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## Skin Lesion Caused by ST398 and ST1 MRSA, Spain<sup>1</sup>

**To the Editor:** Human infections caused by methicillin-resistant *Staphylococcus aureus* (MRSA) sequence type 398 (ST398) have been emerging in recent years in Europe (1,2). Pigs represent a common reservoir of MRSA ST398, and working with these animals may constitute a risk factor for MRSA carriage and possible infection (2–4). We report a case of human infection caused by MRSA ST398 in Spain.

A 12-year-old girl living close to a pig farm, where her father and mother worked, sought treatment for a skin lesion on her chin. Two types of MRSA were recovered from the lesion, and it resolved after topical

treatment with mupirocin over 10 days. MRSA isolates recovered were characterized by multilocus sequence typing (MLST) and by staphylococcal cassette chromosome (SCC) *mec*, *spa*, and *agr* typing as described (3). The presence of genes encoding Pantón-Valentine leukocidin (PVL) (*lukF/lukS*), toxic shock syndrome toxin-1 (*tsst1*), and exfoliative-toxin A and B (*eta*, *etb*) was investigated by PCR (2,3). Antimicrobial susceptibility tests were carried out by using the VITEK-2 system (bioMérieux, Marcy l’Étoile, France), and *mecA*, *ermA*, *ermB*, *ermC*, *mrsA*, *tetK*, *tetL*, *tetM*, *ant(4’)-Ia*, *aph(3’)-III*, and *aph(2’)-aac(6’)* resistance genes were studied by PCR (5). *dfiK* gene detection was performed by using primers designed from the sequence FM207105 included in GenBank (*dfiK*-F 5'-GAGAATCCCAGAGGATTGGG; *dfiK*-R, 5'-CAAGAAGCTTTTCGCTCATAAA), and the obtained amplicons were sequenced for confirmation. Mutations in quinolone targets were determined by sequence analysis of *grrA* and *gyrA* genes (6). In addition, MRSA isolates were typed by pulsed-field gel electrophoresis (PFGE) ([www.harmony-microbe.net/microtyping.htm](http://www.harmony-microbe.net/microtyping.htm)).

One of the clinical MRSA isolates recovered from the lesion and typed as ST398-*spa*-t011 showed resistance to tetracycline, macrolides, and lincosamides and harbored 5 antimicrobial resistance genes. The second MRSA strain was typed as ST1-*spa*-t127 and showed a multiresistance phenotype with 11 resistance genes, as well as the Ser80Phe and Ser84Leu amino acid changes in GrlA and GyrA proteins, respectively.

To establish the MRSA nasal colonization status of the patient and of her relatives (father, mother, and brother, all of whom had contact with

<sup>1</sup>This study was presented as a poster at the 19th European Congress of Clinical Microbiology and Infectious Diseases, Helsinki, Finland, 2009.