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An Influenza AH3 Outbreak in a Hospital, Nara Prefecture, Japan, in Summer 2005

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An influenza outbreak occurred in summer, a non-epidemic season, in a hospital in the northern area of Nara Prefecture, Japan. The outbreak occurred in a ward for mentally and physically disabled patients. On July 15, the first patient, a 43-year-old male inpatient, developed fever (38.7°C) and fatigue of unknown origin. The second patient showed similar symptoms on July 31, and the disease then spread rapidly among inpatients and staff members from August 3 to August 10. A total of 28 inpatients and 10 staff members developed a similar illness (Fig. 1). All the patients showed typical influenza-like symptoms; fever (37.3 to 40.0°C), dyspnea, cough and rhinorrhea. In the hospital, the patients were examined with a rapid influenza detection kit using nasal drips and diagnosed with influenza A virus infection.

To obtain detailed information, we analyzed the virus by the RT-PCR method, DNA sequencing and hemagglutination inhibition (HI) assay. Influenza AH3 virus was detected by the RT-PCR method reported by Shimizu et al. (1), using specific primers (D01 and D02) for hemagglutinin genes of influenza AH3 virus. Next we determined the sequence of 222 nucleotides after TA cloning (pSTBlue-1 AccepTor™ Vector Kit; Novagen, Madison, Wis., USA) and compared the sequence with those of influenza AH3 strains in GenBank using a BLAST search. The results revealed that the detected A/Nara/29/2005 strain shared a high nucleotide homology with influenza A/Wyoming/03/2003 (GenBank accession no. AY531033). Nucleotide and amino acid sequences of the hemagglutinin region are shown in Figures 2A and 2B. Nucleotide and amino acid homologies between these two strains were 98.7% (227/231 bases) and 97.4% (75/77 amino acids), respectively. The A/Nara/29/2005 strain had four point mutations. Three provided amino-acid replacement: 429 (G →

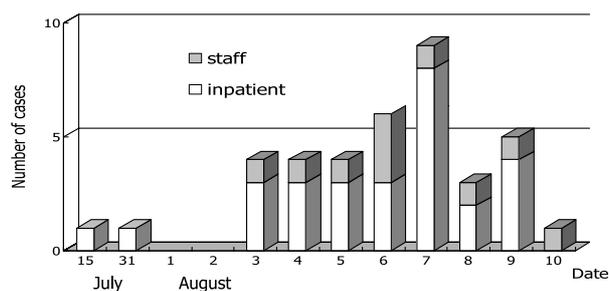


Fig. 1. Case distribution of the nosocomial infection.

A, Ala to Thr), 483 (A → C, Lys to Asn) and 524 (A → T, Tyr to Phe), and one was a mutation with no amino acid change (position No. 444). Virus isolation was attempted from six specimens by inoculating the samples onto MDCK cells. A cytopathic effect was observed in all of the inoculated cells by day 3 or 4 after the inoculation. HI assay was performed using antisera against A/Wyoming/03/2003, A/Kumamoto/102/02 and A/Panama/2007/99, provided by the National Institute of Infectious Diseases, Tokyo. HI titers against respective antisera were 640 (homotypic titer, 1280), 640 (homotypic titer, 1280) and 20 (homotypic titer, 320). The results suggest that the isolated A/Nara/29/2005 strain was nearly identical to A/Wyoming/03/2003, the vaccine strain in the 2004/05 season (2).

In conclusion, the outbreak was caused by influenza AH3 virus, which was identified as an A/Wyoming/03/2003-like virus. This was the main epidemic strain in Nara Prefecture in the 2004/2005 season.

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A

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A/Wyoming/3/03 : CCT TAT GAT GTG CCG GAT TAT GCC TCC CTT AGG TCA CTA GTT GCC TCA TCC GGC ACA CTG
A/Nara/29/2005 :

GAG TTT AAC AAT GAA AGC TTC AAT TGG GCT GGA GTC ACT CAG AAT GGA ACA AGC TCT GCT
. A A

TGC AAA AGG AGA TCT AAT AAA AGT TTC TTT AGT AGA TTG AAT TGG TTG ACC CAC TTA AAA
. C

TAC AAA TAC CCA GCA TTG AAC GTG ACT ATG CCA AAC AAT GAA
T

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B

A/Wyoming/3/03 : PYDVPDYASL RSLVASSGTL EFNNEFNWA GVTQNGTSSA CKRRSNKSFF SRLNWLTHLK YKYPALNVTM PNNE
A/Nara/29/2005 : T N F

Fig. 2. Nucleotide (A) and predicted amino acid (B) sequences of the A/Nara/29/2005 gene (343-564).

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