minimum of 10 d. Patients should be retested for ongoing viral secretion every 5 d and negative results confirmed with a follow-up sample after 48 h. Classic virus isolation in addition to molecular methods may also identify potentially infectious patients.

Prophylactic neuraminidase inhibitor use in such patients also needs to be addressed. Resistance is more likely with the reduced prophylactic dose of oseltamivir and is more likely to be a problem in immunocompromised patients. Zanamivir is now the drug of choice for prophylaxis for such patients, although some experts propose no prophylaxis and instead early treatment after symptom onset (*9*).

Immunocompromised patients are more likely to shed virus for prolonged periods and are more likely to develop oseltamivir-resistance, especially when this drug is used as monotherapy. Further clinical experience and trials will support or refute newer guidelines on the management of pandemic (H1N1) 2009 in such patients.

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Grant A. Hill-Cawthorne, Silke Schelenz, Matthew Lawes, and Samir Dervisevic

Author affiliations: University of East Anglia, Norwich, UK; and Norfolk and Norwich University Hospital, Norwich

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References

1. Dingle KE, Crook D, Jeffrey K. Stable and noncompetitive RNA internal control for routine clinical diagnostic reverse transcription–PCR. J Clin Microbiol. 2004;42:1003–11. DOI: 10.1128/JCM.42.3.1003-1011.2004
2. Centers for Disease Control and Prevention. Oseltamivir-resistant novel influenza A (H1N1) virus infection in two immunosuppressed patients. MMWR Morb Mortal Wkly Rep. 2009;58:893–6.
3. Gooskens J, Jonges M, Claas ECJ, Meijer A, Kroes ACM. Prolonged influenza virus infection during lymphocytopenia and frequent detection of drug-resistant viruses. J Infect Dis. 2009;199:1435–41. DOI: 10.1086/598684
4. World Health Organization. Update on oseltamivir-resistant pandemic A (H1N1) 2009 influenza virus. Wkly Epidemiol Rec. 2010;85:37–48 [cited 2010 Mar 4]. http://www.who.int/wer/2010/wer8506.pdf
5. Weinstock DM, Gubareva LV, Zuccotti G. Prolonged shedding of multidrug-resistant influenza A virus in an immunocompromised patient. N Engl J Med. 2003;27:867–8. DOI: 10.1056/NEJM200302273480923
6. Moscona A. Neuraminidase inhibitors for influenza. N Engl J Med. 2005;353:1363–73. DOI: 10.1056/NEJMra050740
7. Health Protection Agency. Inpatient clinical management issues relating to oseltamivir-resistant pandemic (H1N1) 2009 influenza virus. Version 2, 2010 [cited 2010 Mar 4]. http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1259152289698
8. World Health Organization. Oseltamivir resistance in immunocompromised hospital patients. Pandemic (H1N1) 2009 briefing note 18 [cited 2010 Apr 13]. http://www.who.int/csr/disease/swineflu/notes/briefing_20091202/en/index.html
9. World Health Organization. Oseltamivir-resistant pandemic (H1N1) 2009 influenza virus, October 1999. Wkly Epidemiol Rec. 2009;84:453–68 [cited 2010 Mar 4]. http://www.who.int/wer/2009/wer8444.pdf

Address for correspondence: Samir Dervisevic, Virology Centre, Department of Microbiology, Norfolk and Norwich University Hospital, Bowthorpe Rd, Norwich NR2, UK; email: samir.dervisevic@nnuh.nhs.uk

Sapovirus in Adults in Rehabilitation Center, Upper Austria

To the Editor: Contrary to norovirus (NoV) infections, sapovirus (SaV) is believed to affect mainly young children (*1*), although recent studies show that SaV is present in all age groups (*2*,*3*). SaV has been classified into 5 genogroups, of which GI, GII, GIV, and GV affect humans (*4*). SaV can be transmitted in various ways, including person-to-person fecal–oral route, by aerosol, and by consumption of contaminated food or water (*5*). Outbreaks of SaV have been reported in various settings but are less common than NoV outbreaks (*1*,*6*).

During October 2–7, 2008, an outbreak of gastroenteritis occurred in a rehabilitation center in Upper Austria. Signs including diarrhea, vomiting, and fever developed in 21 adult patients and 12 staff members. The observed signs and the likely incubation period initially suggested NoV as the cause of the outbreak. Stool specimens of 10 patients were collected and submitted to the Institute for Medical Microbiology and Hygiene in Graz.

Along with microbiologic investigations, infection control measures were introduced by local authorities on each affected ward. The earliest reported onset of illness was on October 2, 2008, in a 52-year-old woman on the third floor. The next day 2 additional patients on the same floor and 1 member of the kitchen staff showed symptoms. Another 7 patients, on 3 different floors, and 2 of the medical staff suffered from symptoms the following day (October 4). The outbreak peaked with 11 cases 3 days after the initial episode of vomiting. An additional 9 persons became infected (5 patients, a doctor, janitor, psychotherapist, and kitchen worker) within the following 2 days. The affected patients were pre-

dominantly elderly; mean age was 54 years (range 20–81 years, male:female ratio 1:0.83). Clinical signs continued for 24 hours.

Routine microbiologic cultures for enteric bacterial pathogens were performed and showed negative results. A NoV-specific 1-tube real-time PCR assay (LightCycler 2.0; Roche Applied Science, Mannheim, Germany) with primers/probes reported by Hoehne et al. (*7*) and ELISAs (Ridascreen; R-Biopharm AG, Darmstadt, Germany) were conducted to detect rotavirus and adenovirus antigen. All tests yielded negative results. Subsequently, 5 of 10 samples were submitted to the Robert Koch Institute in Berlin for further investigation. SaV was identified in 4 samples by using the reverse transcription–PCR described by Oka et al. (*8*); mean viral load was 2.65×10^8 RNA copies/g stool (range 3.3×10^7–7.7×10^8 copies/g stool). Direct sequencing of the appropriate amplification product showed 100% identical nucleotide sequences, which indicated 1 causative strain.

Retrospective testing of the 10 specimens showed 9 SaV-positive samples. Subsequently, genotyping was conducted by using a 1,130-bp amplification product of the polyprotein gene open reading frame 1 spanning the recombination site at the junction between the polymerase gene and the capsid gene. One specimen was amplified by RT-nested PCR with sense primer SV 53a (5′-TAGACTACAGCAAGTGGGA-3′, nt position 4356–4374), antisense primer SV 63 (5′-ACACCATGT TGGACACGCTGC-3′, nt position 6901–6881), and SuperScript III One-Step RT-PCR System with Platinum Taq High Fidelity (Invitrogen, Paisley, UK) for the first-round PCR. For the second-round PCR, the HotStarTaq Master Mix Kit (QIAGEN, Hilden, Germany) and primer SV 55a (5′-CCMTCKGGCATGCCATTCAC-3′, nt position 4529–4548) and SV60 (5′-ATGTTAAATGTGATAGGATCCA C-3′, nt position 5658–5636) were used (nucleotide positions according to GenBank accession no. DQ058829).

Phylogenetic analysis of the second-round PCR product (GenBank accession no. GU724600) showed 97% nucleotide identity to the strain Angelholm/SW278/2004/SE (GenBank accession no. DQ125333), which is a known intergenogroup recombinant virus (II.2/ IV) as seen in Japan and Sweden (*9*). Thus, by sequence analysis of the polymerase region, our strain Graz1561/2008/Austria was grouped into genogroup II; the capsid region belonged to genogroup IV.

Diarrhea and vomiting were the most common signs in patients, 97% and 73%, respectively. Fever was recorded for only 1 case-patient. Our findings are consistent with those of SaV outbreak studies in adults reported by Johansson et al. (*3*), who reported diarrhea in 72% and vomiting in 56% of the case-patients they studied.

The new genetic background of the recombinant virus may have enhanced host susceptibility by evading the immune response and is therefore able to affect adults. Our study shows that SaV causes outbreaks of gastroenteritis in adults; consequently, the role of SaV in the adult population should be reconsidered. We suggest that diagnostics for SaV should be included in the study of gastroenteritis outbreaks in adults, especially when clinical signs suggest NoV as the causative agent but no diagnostic confirmation can be achieved.

Claudia Mikula, Burkhard Springer, Sandra Reichart, Katharina Bierbacher, Alfred Lichtenschopf, and Marina Hoehne

Author affiliations: Austrian Agency for Health and Food Safety, Graz, Austria (C. Mikula, B. Springer, K. Bierbacher); Public Health Authority Upper Austria, Linz, Austria (S. Reichart); Rehabilitation Center Weyer, Weyer, Austria (A. Lichtenschopf); and Robert Koch-Institute, Berlin, Germany (M. Hoehne)

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References

1. Pang XL, Honma S, Nakata S, Vesikari T. Human caliciviruses in acute gastroenteritis of young children in the community. J Infect Dis. 2000;181(Suppl 2):S288–94. DOI: 10.1086/315590
2. Pang XL, Lee BE, Tyrrell GJ, Preiksaitis JK. Epidemiology and genotype analysis of sapovirus associated with gastroenteritis outbreaks in Alberta, Canada. J Infect Dis. 2004–2007;2008:19.
3. Johansson PJ, Bergentoft K, Larsson PA, Magnusson G, Widell A, Thorhagen M, et al. A nosocomial sapovirus-associated outbreak of gastroenteritis in adults. Scand J Infect Dis. 2005;37:200–4. DOI: 10.1080/00365540410020974
4. Hansman GS, Oka T, Sakon N, Takeda N. Antigenic diversity of human sapoviruses. Emerg Infect Dis. 2007;13:1519–25.
5. Hansman GS, Sano D, Ueki Y, Imai T, Oka T, Katayama K, et al. Sapovirus in water, Japan. Emerg Infect Dis. 2007;13:133–5. DOI: 10.3201/eid1301.061047
6. Ike AC, Hartelt K, Oehme RM, Brockmann SO. Detection and characterization of sapoviruses in outbreaks of gastroenteritis in southwest Germany. J Clin Virol. 2008;43:37–41. DOI: 10.1016/j.jcv.2008.04.003
7. Hoehne M, Schreier E. Detection of norovirus genogroup I and II by multiplex real-time RT-PCR using a 3′-minor groove binder-DNA probe. BMC Infect Dis. 2006;6:69. DOI: 10.1186/1471-2334-6-69
8. Oka T, Katayama K, Hansman GS, Kageyama T, Ogawa S, Wu FT, et al. Detection of human sapovirus by real-time reverse transcription-polymerase chain reaction. J Med Virol. 2006;78:1347–53. DOI: 10.1002/jmv.20699
9. Hansman GS, Takeda N, Oka T, Oseto M, Hedlund KO, Katayama K. Intergenogroup recombination in sapoviruses. Emerg Infect Dis. 2005;11:1916–20.

Address for correspondence: Claudia Mikula, Österreichische, Agentur für Gesundheit und Ernährungssicherheit GmbH, Institut für Medizinische Mikrobiologie und Hygiene, Beethovenstrasse 6, Graz A-8010, Austria; email: claudia.mikula@ages.at