Laboratory and epidemiology communications

A Food Poisoning Outbreak Due to Food Handler-Associated Contamination with the Human Norovirus GII.P16-GII.2 Variant Strain in Italian Cuisine in Tokyo during the 2016/17 Winter Season

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Human norovirus (HuNoV) is a major causative agent of gastroenteritis and food poisoning worldwide (1). To date, over 30 genotypes of HuNoV have been confirmed (1). Furthermore, each genotype of HuNoV has evolved uniquely, resulting in many variant strains, even within the same genotype (2). For example, GII.4 strains have frequently generated variant strains (i.e., GII.4v 2006, Sydney strain), leading to pandemics of gastroenteritis in many countries, including Japan (3, 4). Japanese national surveillance data (Infectious Diseases Weekly Report) on gastroenteritis have indicated that HuNoV GII.2 strains were the third or fourth most prevalent genotypes over the past 5 winter seasons (5). However, the Infectious Agents Surveillance Report showed that in the 2016/17 winter season in Japan GII.2 strains emerged suddenly and caused large outbreaks of acute gastroenteritis among children (6). Very recent reports from Germany, China, and Taiwan have also shown that the GII.P16-GII.2 variant (GII.2v) is a prevalent genotype in patients with viral gastroenteritis (7–9). Here, we encountered a large food-poisoning outbreak due to consumption of Italian cuisine with secondary contamination by GII.2v via food handlers in a restaurant.

A public health office in the Tokyo metropolitan area received a report that some guests who had attended a party at a restaurant held on December 11, 2016 showed symptoms such as diarrhea and vomiting beginning on December 12, 2016. An epidemiological investigation by jurisdictional public health officials revealed that many other guests who had attended the party at the same restaurant on December 11, 2016 also showed acute gastroenteritis symptoms. A total of 136 guests who attended the same party at the restaurant during December 12 to 14, 2016. The mean incubation period was 34.3 h (34.3 ± 11.1 h, mean ± standard deviation). The epidemic curve showed that the highest number of patients developed acute gastroenteritis on December 13, 2016. The median age of patients was 48 years (range, 6–62 years). There were 47 males and 29 females (gender ratio of 1.6:1, respectively). They showed typical symptoms of gastroenteritis, including diarrhea (77.6%), nausea (76.5%), malaise (65.2%), fever (60.5%), and vomiting (57.9%). Moreover, 11 employees (food handlers and service staff) working in the restaurant developed acute gastroenteritis on December 12, 2016. We collected fecal specimens from patients, food handlers, and service staff after receiving their oral informed consent in compliance with the Food Sanitation Act. Personal data of these patients were anonymized. We collected fecal samples from 76 guests who consumed Italian cuisine at the restaurant on December 11, 2016 and 37 employees of the restaurant. To detect HuNoV, we used real-time reverse transcription-polymerase chain reaction. The NoV GII strain was detected in 67 of the 76 patients (88.2%) and 13 of the 37 employees (35.1%, 4 food handlers and 9 service staff). Moreover, we sequenced the RdRp and VP1 coding regions of the present NoV strains detected from 5 patients and 5 employees and constructed phylogenetic trees by the maximum likelihood method. The obtained nucleotide sequences in the present study were deposited in GenBank under the accession numbers LC279234 to LC279243. The nucleotide sequences of the RdRp (1530 nt) and VP1 (1626 nt) coding regions almost completely matched among the strains studied [99.93% (only one nucleotide substitution) and 100.0% identical, respectively]. The phylogenetic trees estimated that these strains were GII.2v (Fig. 1). Based on these results, we estimated that the food poisoning was due to food-handler-associated contamination by GII.2v in the Italian cuisine. Among them, 76 guests developed acute gastroenteritis from December 12 to 14, 2016. The mean incubation period was 34.3 h (34.3 ± 11.1 h, mean ± standard deviation). The epidemic curve showed that the highest number of patients developed acute gastroenteritis on December 13, 2016. The median age of patients was 48 years (range, 6–62 years). There were 47 males and 29 females (gender ratio of 1.6:1, respectively). They showed typical symptoms of gastroenteritis, including diarrhea (77.6%), nausea (76.5%), malaise (65.2%), fever (60.5%), and vomiting (57.9%). Moreover, 11 employees (food handlers and service staff) working in the restaurant developed acute gastroenteritis on December 12, 2016. We collected fecal specimens from patients, food handlers, and service staff after receiving their oral informed consent in compliance with the Food Sanitation Act. Personal data of these patients were anonymized. We collected fecal samples from 76 guests who consumed Italian cuisine at the restaurant on December 11, 2016 and 37 employees of the restaurant. To detect HuNoV, we used real-time reverse transcription-polymerase chain reaction. The NoV GII strain was detected in 67 of the 76 patients (88.2%) and 13 of the 37 employees (35.1%, 4 food handlers and 9 service staff). Moreover, we sequenced the RdRp and VP1 coding regions of the present NoV strains detected from 5 patients and 5 employees and constructed phylogenetic trees by the maximum likelihood method. The obtained nucleotide sequences in the present study were deposited in GenBank under the accession numbers LC279234 to LC279243. The nucleotide sequences of the RdRp (1530 nt) and VP1 (1626 nt) coding regions almost completely matched among the strains studied [99.93% (only one nucleotide substitution) and 100.0% identical, respectively]. The phylogenetic trees estimated that these strains were GII.2v (Fig. 1). Based on these results, we estimated that the food poisoning was due to food-handler-associated contamination by GII.2v in the Italian cuisine. 

GII.2v emerged suddenly during the 2016/17 winter season in Japan and other countries (6–9). The strains also caused large outbreaks of acute gastroenteritis in
children in various countries (6–10), and these may have been closely related genetically (10). However, detailed genetics and antigenicity of these are not exactly known at present (10). Thus, additional analyses, including molecular epidemiology of the strains, may be needed.

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Conflict of interest None to declare.

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