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Outbreak of Enterohemorrhagic Escherichia coli O26 in Niigata City, Japan

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On September 2, 2006, a clinic notified the Niigata City Health Center of two cases of enterohemorrhagic Escherichia coli O26 VT1-positive (EHEC O26 VT1) infection. Acting on this information, the health center immediately started an investigation including sampling swab specimens from the patients’ homes, collecting stool specimens from family members and their contacts, and other necessary epidemiological examinations. The patients’ food consumption history revealed that both patients dined at the same restaurant A. Swab specimens from the restaurant, specimens of food stored in the restaurant, and the employees’ stool specimens were also collected.

The results of the investigation are summarized in Figure 1. One specimen of giara (bovine fourth stomach or abomasum), a cook’s stool specimen, and those of the patient’s and his four companions who dined together were positive for EHEC O26 VT1.

The Niigata City Public Health Center suspected an outbreak, and in order to prevent an expansion of EHEC O26 infection, a telephone consultation system was conducted on September 6 and 7. Pathogen carriers #7 to #13 were found by this service. The clinical symptoms of these carriers were slight or almost absent. Carriers #7, #8, and #13 ate a meal in restaurant A on August 26, and carriers #9 to #12 shared food purchased at restaurant A. Carriers #9 and #10 and carriers #11 and #12 were brothers, and they went to nursery schools B and C, respectively. Investigation of stool specimens of the employees and children in the nursery schools revealed one nursery school child, patient #14, was positive for the bacteria. On September 20, secondary infection in a family, case #15, was detected. A total of 16 cases were detected. The pathogen-positive giara was, however, not part of the food served on August 26, the presumed day of the patients’ exposure to the pathogen, but was purchased later on August 31 and chopped and stored.

A total of 471 stool specimens, 40 swab specimens, and 10 samples of broiled meat were used for the isolation and identification of pathogens. The selection medium was Cefixime-potassium tellurite-added Rhamnose MacConkey plates. Most stool specimens showed dominant growth of EHEC O26 VT1. Specimens from patients #13 and #15, however, produced mixed cultures of EHEC O26 VT1 and commensals, and the colonies were small at 18 h of incubation with an ordinary colony size being attained at 24 h or later. For food speci-
mens, the bacteria were isolated only from the giara after amplification in a novobiocin-added modified *E. coli* medium. Direct isolation from other food specimens was attempted but was negative even after amplification using 10% suspension in an amplification medium prepared from 10 g of the specimens.

Pulsed-field gel electrophoresis (PFGE) pattern was identical for all the specimens (Fig. 2). The PFGE pattern of specimen F from the giara and #13 differed from the others by one band, but were otherwise similar to the others. Therefore, the outbreak was probably caused by a common infection source.

Epidemiologically, the infection source was the food served at restaurant A on August 26. However, it was difficult to identify the dish that caused the infection, because the giara that was found pathogen-positive came to the restaurant after August 26. Thus, it could not have been the infection source. The cook in restaurant A who was found positive for the pathogen could have been responsible, but we cannot rule out the possibility that he was somehow infected on the same day.

In this outbreak, the patients were restricted to those who had a chance to have meals served to them in restaurant A on August 26, and no other patients were reported. The outbreak terminated when the symptomatic patients stopped excreting the pathogen about a month later. One of the characteristics of EHEC O26 VT1 is frequent secondary infection such as to family members, and the present case was not an exception.

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