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On March 31, student health services at a university in the District of Columbia (DC) notified the DC health department that an increased number of students had become ill with acute gastroenteritis beginning March 29. Some ill students reported eating tuna or chicken salad sandwiches from dining hall A on campus. On March 31, the DC health department initiated an outbreak investigation. This report summarizes results of the investigation, which indicated that group A rotavirus transmitted by food was the cause of the outbreak.

Telephone interviews were conducted with students who reported illness to student health services, with additional ill students who were identified during interviews, and with healthy controls selected randomly from the university registry of students residing on campus. A case of gastroenteritis was defined as three or more episodes of diarrhea and/or two or more episodes of vomiting within a 24-hour period in a student with onset on or after March 20. Controls and case-patients whose illness onset occurred during March 27--31 were questioned about food history, residence and dining hall, source of water, use of a public access computer or sports equipment at the university gym, and attendance at social or athletic events. Electronic records of student meal attendance were available for 49 case-patients with illness onset during March 27--31 and for 55 control subjects.

FIGURE 1. Number* of gastroenteritis† cases among college students, by date of illness onset — District of Columbia, March 27–April 11, 2000

* n=55.
† A case of gastroenteritis was defined having three or more episodes of diarrhea and/or two or more episodes of vomiting within a 24-hour period in a student with onset on or after March 20.
Twenty-three (79%) of 29 employees of dining hall A were interviewed to identify their work duties and determine whether they were ill. Stool specimens were collected during March 29--April 10 from six ill students and 21 dining hall A employees. Samples were screened for bacterial and parasitic pathogens at a commercial laboratory and for viral pathogens at CDC.

The outbreak among students began March 27 and peaked at 19 cases on March 31 (Figure 1). A total of 108 students (55 were identified by telephone interviews and 53 were self-reported) had gastrointestinal symptoms during March 26--April 11; 85 (79%) had illness that met the case definition. The attack rate among students residing on campus was 5% (77 of 1641), with no significant differences in attack rates by sex, occupancy of residence hall, or grade level. Eight case-patients resided off campus (attack rate: 0.02%). Among the 83 case-patients for whom a complete list of symptoms was reported, 77 (93%) had diarrhea, 75 (90%) abdominal pain or discomfort, 69 (83%) loss of appetite, 67 (81%) nausea, 64 (77%) fatigue, 56 (67%) vomiting, 49 (59%) headache, 48 (58%) chills, 48 (58%) subjective or low-grade fever, and 42 (51%) myalgia. Sore throat, cough, and/or congestion were reported by six case-patients with onsets on or after April 2. The median duration of illness was 4 days (range: 1--8 days). Nine (11%) case-patients received intravenous fluids to treat dehydration.

Of those who completed the telephone interview, 40 (91%) of 44 case-patients and 27 (68%) of 40 controls ate at least one deli sandwich from campus dining hall A during March 27--30 (p=0.017; odds ratio [OR]=4.8; 95% confidence interval [CI]=1.3--22.1). During March 27--30, four (8%) of 49 case-patients ate four or more meals at dining hall B compared with 18 (33%) of 55 controls (p=0.005; OR=0.2; 95% CI=0.04--0.6). Food histories of employees were not recorded; however, six employees reported illness.

Stool specimens of students and employees were negative for bacterial and parasitic pathogens and for Norwalk-like viruses. Using electron microscopy, enzyme immunoassay, and reverse transcriptase-polymerase chain reaction (RT-PCR), nine (33%) of 27 specimens were positive for group A rotavirus. Rotavirus positive stool specimens from four students and three employees were identified as genotype combination P[4],G2 by RT-PCR. Two of the three P[4],G2-positive employees were line cooks who reported having symptoms of gastroenteritis on March 27 and April 2, respectively, while the third positive employee, a deli server, reported no illness.
Editorial Note:

Group A rotavirus is the most common cause of childhood diarrhea worldwide, infecting >90% of children by age 3 years (1). Because rotavirus immunity develops early in life, disease among older children and adults is uncommon (1). Although the role of rotavirus in diarrhea outbreaks in adults has not been well studied, it has been documented as the cause of adult diarrheal outbreaks in hospitals (2), nursing homes (3), isolated communities (4), and in travelers (5). Also, parents of children infected with rotavirus have been reported to experience acute gastroenteritis (6). However, the rotavirus G and P protein-type combinations, the proteins that elicit an immune response in humans, were not characterized in most of these reports.

The rapid increase and gradual decline of the campus outbreak suggest that the infection was foodborne during the first week and was spread person-to-person during the following week. During the first week, illness was associated with eating sandwiches at dining hall A and was associated inversely with eating frequently at dining hall B. The employee who prepared sandwich fillings did not report illness and tested negative for rotavirus. None of the three deli servers who assembled and served sandwiches reported illness; however, one was rotavirus P[4],G2 positive. It is unknown whether the deli server who tested positive was infected before the outbreak among students.

This rotavirus serotype G2 outbreak was unusual for two reasons; food was implicated as the source of infection and the adults affected should have been immune. During April 2000, a gastroenteritis outbreak among adults in Japan also was caused by foodborne transmission of group A rotavirus serotype G2 (7). These adults should not have been susceptible to severe rotavirus illness. G2 strains often are found combined with serotype P[4]1B (8). The G and P neutralization antigens of serotype G2 strains may allow G2 strains to escape immunity induced by the more common G1, G3, and G4 strains. In addition, G2 has been associated with more severe dehydration during diarrheal episodes.
in children than other common strains (9). These outbreaks of rotavirus gastroenteritis in adults in the United States and Japan raise questions about the persistence of immunity to rotavirus and the virulence of G2 strains. Investigators and clinicians should consider rotavirus as a possible cause of acute gastroenteritis in adults.

References


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**Case 1 Questions:**

1. These are the elements of defense systems. Complete this table (re-type it to make more space) by listing specific examples in the right hand column.
Elements of (Immune) Defense System

<table>
<thead>
<tr>
<th>Barriers to Infection</th>
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<tbody>
<tr>
<td>Surveillance/Pathogen Detection</td>
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<tr>
<td>Discrimination (Self vs. Non-self)</td>
</tr>
<tr>
<td>Troops</td>
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<tr>
<td>Troop recruitment and training</td>
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<tr>
<td>Communication/Regulation</td>
</tr>
<tr>
<td>Weapons/Effectors</td>
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<tr>
<td>Pathogen Destruction</td>
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<tr>
<td>Tissue Repair</td>
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<tr>
<td>Immune Memory</td>
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2. List the information in the case about the pathogen (rotavirus) and the disease (gastroenteritis). Include in your list how the pathogen is transmitted, where it enters the body, what the disease symptoms are, how the disease is diagnosed, and any treatments given. Make a separate list of terms with which you are unfamiliar so we can define them in class.

3. What immunological barriers must the rotavirus overcome to infect someone (HINT: remember where the virus enters the body)?

4. What is immunity? To what can people become immune? How long does immunity last? Did the students described in the case have immunity to rotavirus (how do you know)?

5. For each of the following leukocytes (white blood cells, WBC) list at least one function in host defense: neutrophil, eosinophil, mast cell, macrophage (Mφ), dendritic cell (DC), B cell, T cell, Natural Killer (NK) cell. What is the function of M cells in the mucosal immune system?

6. What are the primary and secondary lymphoid organs? Name specific examples and say what their purpose is. How do leukocytes move around the body?

7. Compare and contrast innate and adaptive (acquired) immunity in terms of speed, specificity, and memory.
Rotavirus is a complex virus with a double-stranded 11-segment RNA genome and proteins named VP 1-7 (structural proteins) and NSP 1-5 (which include RNA-dependent RNA-polymerases and regulatory proteins; see above diagram.) Viruses with this kind of structure bind to host cells using the protein on the spikes (in this case VP4) and are taken into the cell by endocytosis. Rotavirus binds to and infects intestinal epithelial cells (enterocytes) responsible for secreting digestive enzymes (disaccharidases and peptidases) and absorbing nutrients and water (Ramig, 2004). The virus RNA is uncoated inside the enterocyte and replicates in the enterocyte cytoplasm. New virions (virus particles) are formed by assembling the virus RNA and structural proteins; when enough new virions accumulate in the host cytoplasm, the host cell lyases and the virions are released to enter new cells. A recent finding is that NSP-4 is released from the enterocyte and acts as an enterotoxin (Morris and Estes, 2001). Rotaviruses are shed in the feces at $10^9$ /ml; as few as 10-100 virions are required for infection (Carter, 2005).

7. The molecules "seen" by the immune system are called antigens. What are the rotavirus antigens (see diagram above) that the immune system will see? What is a neutralizing antigen? Is is a coincidence that the neutralizing antigens VP4 and VP7 are on the outside of the virus?

8. Name the recognition molecules (antigen receptors) on T cells and B cells. How are they different in their ability to recognize (bind) antigen? How is it that the immune system can recognize so many different antigens but not (usually) self antigens?

9. Based on the information in Chapter 1, how will the immune system respond to this infection? List each immune effector and its function as bullets. (For example, will macrophages be involved? If so, what will they do and where? Will there be inflammation? Where will antibodies be produced? What will T cells do?).

Case adapted and Modified from Dr. Janet Deckert at the University of Arizona. Her email is jdeckert@u.arizona.edu and her website is webMic419 Home
Supplementary Materials:

**Infectious Disease, Immunity Rules, Antigen, ToolBox**