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ORIGINAL ARTICLE

Clinical manifestations of a cluster of rotavirus infection in young infants hospitalized in neonatal care units

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Purpose: To define the clinical manifestations of rotavirus (RV) infection in neonates and young infants hospitalized in neonatal care units, which are rarely reported.
Materials and Methods: From October 2008 to September 2010, a total of 153 stool specimens positive for RV were detected from 100 neonates and young infants hospitalized in neonatal care units of our hospital. Four infants had two episodes of RV infection. Demographics and clinical presentations of these infants were collected and analyzed. The infants were further classified as having hospital-acquired (HA) or community-acquired (CA) RV infection.
Results: Of the 104 episodes from 100 patients, 76 (73%) were classified as HA. Fifty-six infants were male. The mean age of onset was 2 days. The most common presentations were loose stool passages (52.9%), abdominal distension (51.9%), blood or mucus in stool (42.3%), and unstable vital signs (32.7%). Watery character in stool passage was identified in 13.5% of the infants and vomiting in 21.2%. A picture suggestive of necrotizing enterocolitis (NEC) was identified in 22 episodes (21.1%), and 12 of these were stage II or above. The average number of hospitalization days from the onset of HA-RV infection was 23 days. Compared with those in

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the CA group, the infants in the HA group had a significantly higher rate of blood or mucus in stools (52.6% vs. 14.3%, $p < 0.01$) and unstable vital signs (39.5% vs. 14.3%, $p = 0.02$), but a lower rate of watery diarrhea (9.2% vs. 28.6%, $p = 0.04$) and fever (13.8% vs. 42.9%, $p < 0.01$). Overall, there were five deaths, but all of these infants had major diseases.
Conclusion: Bloody, mucoid stools and unstable vital signs, instead of fever with watery diarrhea, are commonly seen in neonates and young infants with RV infection. A substantial proportion of these infants may present as NEC. Once introduced, RV appears to become a troublesome problem of HA infections in neonatal care settings.

Introduction

Rotavirus (RV) is a common pathogen of acute gastroenteritis, particularly in children less than 5 years of age.[1] Disease tends to be most severe in patients 3—24 months of age, although 25% of the cases of severe disease occur after 2 years of age, with serologic evidence of infection developing in virtually all children by 4—5 years of age.[1] Rotavirus infection is most common in winter months in temperate climates,[1] while in Taiwan, RV gastroenteritis occurs throughout the year, with variable seasonal peaks reported.[2—4] The typical manifestations of gastroenteritis caused by RV infection are mild-to-moderate fever with vomiting, followed by the onset of frequent, watery stools. Vomiting and fever typically abate during the second day of illness, but diarrhea often continues for 5—7 days. The stool is usually without gross blood or white blood cells (WBC).[1] Infections in neonates are generally mild or asymptomatic,[1,5] but some may present as necrotizing enterocolitis (NEC),[6] a severe form of gastrointestinal disease in neonates and young infants. Reports of rotaviral infections in this young population have been scanty.

In Chang Gung Memorial Hospital, a tertiary medical center situated in northern Taiwan, a total of 2443 episodes of documented rotaviral infections less than 15 years of age were admitted to the pediatric wards in the past 6 years from 2005 to 2010. The peak season of RV infections was late winter/early spring from February to April in each year (Fig. 1). In contrast, the case number of RV infections hospitalized in neonatal care units was very low except in 2009 and the seasonal distribution was relatively even, without an apparent peak, in each year, suggesting different disease patterns. In 2009, we cared for a large number of babies with documented RV infections in the neonatal care units and found that the clinical features in this age population were different from those in older infants and children. Therefore, we conducted this study to delineate the clinical manifestations of this cluster of RV infections in young infants which are rarely reported in Taiwan.

Materials and methods

From October 2008 to September 2010, stool specimens for RV antigen were analyzed using an enzyme immunoassay (RIDASCREEN® Rotavirus, R-Biopharm AG, Germany) in Chang Gung Memorial Hospital. This commercial kit was reported to exhibit excellent sensitivity (100%), specificity (99.73%), positive predictive value (93.74%), and negative predictive value (100%).[7] A total of 153 stool samples positive for RV were detected from 100 neonates and young infants hospitalized in neonatal care units at Chang Gung Memorial Hospital, a tertiary medical center situated in northern Taiwan. We retrospectively collected the clinical

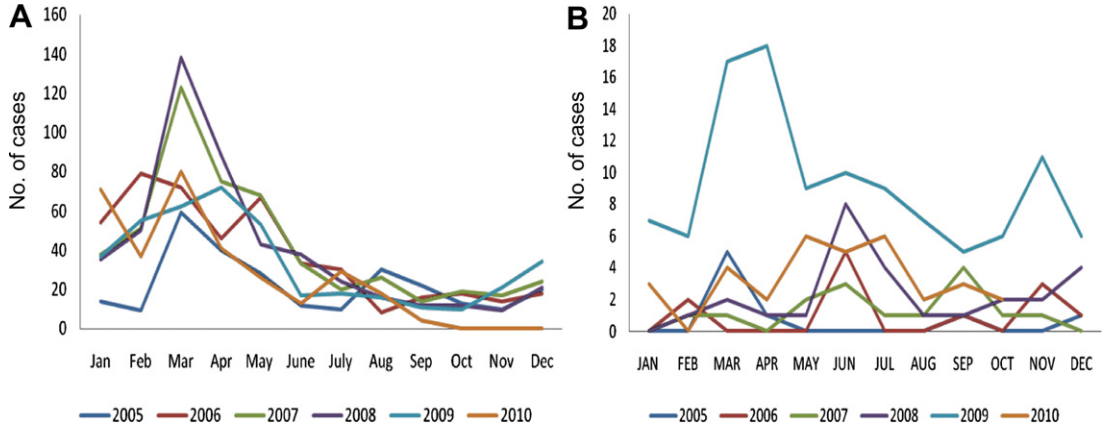

Figure 1. Seasonal distribution of rotavirus infection in the general pediatric wards (A) and neonatal care units (B) at Chang Gung Memorial Hospital during 2005 to 2010.[a] Rotavirus infection was most common from February to April in general pediatric wards, which is similar to the incidence seen in temperate countries in the winter and early spring. [b] The curve of seasonal distribution in neonatal care units did not parallel the distribution in the community.

data, including age, sex, gestational age, birth body weight, clinical manifestations, laboratory findings, and outcomes from these patients. According to the site of acquisition of RV, the patients were categorized as community-acquired (CA) infection and hospital-acquired (HA) infection; the HA group was further classified into three subgroups by gestational ages: full term (gestational age (GA) $>$ 37 weeks), near term (GA between 34 and 37 weeks), and preterm (GA $<$ 34 weeks).

Definitions

Hospital-acquired infection was defined as the onset of symptoms more than 72 hours after admission, or within 72 hours of discharge from hospitals or nursery settings[8]; All other cases were defined as CA. Fever was defined as the body temperature $\geq$ 38 °C. Unstable vital signs consisted of any one of the following symptoms and signs: hypothermia (core temperature $<$36 °C), apnea, bradycardia ($<$ 100 beats per minute), tachycardia ($>$160 beats per minute), desaturation, or hypotension recorded in the medical charts. Recurrence in the same patient was defined as the stool specimens being negative for RV antigen at least twice, with more than a 2-week interval between two episodes. With regard to physiological dehydration of newborn, body weight loss was defined as weight loss over 10% only.

Leukocytosis was defined as a leukocyte count $\geq$34,000/mm^3 for patients aged within 1 week; $\geq$19,500/mm^3 for patients aged between 1 week and 1 month; and $\geq$17,500/mm^3 for patients aged between 1 month and 1 year.[9] Leucopenia was defined as a leukocyte count $\leq$5000/mm^3 in all patients. Thrombocytopenia was defined as a platelet count $<150 \times 10^3$/L. Thrombocytosis was considered when a platelet count was $>400 \times 10^3$/L.

Urinary tract infection was considered when urinalysis revealed pyuria (WBC $>$ 30/μL) with a positive urine culture (one species, colony count $\geq 10^5$ CFU/mL). Metabolic acidosis was recorded when a blood gas revealed base deficit $>$5.0 mEq/L,[9] or if the patient required sodium bicarbonate infusion. Respiratory failure was defined as when the patient deteriorated to require ventilation (including a nasal continuous positive airway pressure, nasal conventional ventilator, or intubation) during their illness. Co-infections included documented urinary tract infection, documented pneumonia (progression on chest radiograph with evidence of sputum culture), documented bacteremia, documented stool culture, or documented viral culture. If the symptoms and signs met the criteria of systemic inflammatory response syndrome,[9] sepsis was recorded. Necrotizing enterocolitis was defined and classified using the modified Bell Staging for Necrotizing Enterocolitis.[10] Coagulopathy was defined as thrombocytopenia and prolonged international normalized ratio (INR) that required platelet and fresh frozen plasma (FFP) transfusion clinically.

Statistical analysis

Data were collected on forms, and were further computerized and analyzed using SPSS software, version 17.0 (SPSS Inc., Chicago, Illinois, USA). Categorical and continuous variables between HA RV infections and CA RV infections were assessed by the Chi-square test and *t* test, respectively. Multiple comparisons of RV infections between full term, near term, and preterm were assessed by using Scheffe's test. A result was considered statistically significant if its two-tailed *p* value was $<$0.05.

Results

During the study period, a total of 104 episodes of RV infection were identified from 100 neonates and young infants. Four infants had two episodes. Fifty-six infants were male. The mean age of onset was 29 days (Table 1). Seventy-six episodes (72.4%) were classified as HA infections. The demographics and clinical manifestations are illustrated in Table 1. The most frequent clinical manifestations were loose stool passages (52.9%), abdominal distension (51.9%), blood or mucus in stool (42.3%), unstable vital signs (32.7%), and feeding intolerance (15.4%). Necrotizing enterocolitis (NEC) was identified in 22 episodes; 10 belonged to Bell's stage I, 10 belonged to stage II, and two belonged to stage III. Compared with those in the CA group, the infants in the HA group had a significantly lower birth weight, lower gestational ages, more underlying diseases, a higher frequency of bloody mucoid stools, and unstable vital signs (Table 1), while fever and watery stool passage were significantly more commonly seen in infants in the CA group.

Co-infections were identified in 16 episodes (18.3%), and included pneumonia in two episodes (*Serratia marcescens* and *Enterobacter cloacae*, respectively), bacteremia in nine (coagulase-negative staphylococci for four, *Acinetobacter baumanii*, *Pseudomonas aeroginosa*, *Viridans Streptococcus*, *Escherichia coli*, and *Enterobacter cloacae* for one each), urinary tract infection in two (*Escherichia coli* for both), *Salmonella* enterocolitis, norovirus gastroenteritis and respiratory syncytial virus infection in one each. Other complications included metabolic acidosis (8.7%), sepsis (7.7%), ileus requiring rectal tube insertion for decompression (2.9%), respiratory failure (3.8%) and death (4.8%) (Table 1).

For laboratory findings, complete blood cells showed leukocyte counts were within the normal range for most patients. There was a large variation of serum C-reactive protein levels, ranging from 0.2 to 164 mg/L. Stool examination revealed positive occult blood in nearly 70% of the patients, and positive pus cell in 15%. Compared with those in the HA group, the infants in the CA group had a significantly lower rate of thrombocytopenia, but higher rate of hyperkalemia and base deficit (Table 2). Among the infants with HA infections, fever and watery stool passage were significantly more commonly seen in term infants than in preterm infants, while preterm infants had a significantly higher frequency of unstable vital signs and NEC (Table 3).

Four patients had recurrences. One of the second episodes was classified as CA and the other three as HA. There were five deaths in these 104 episodes. All of them had underlying major diseases. The first case was a premature infant, with a gestational age (GA) of 24 weeks and patent ductus arteriosus status after surgical ligation and

Table 1 Demographics and clinical manifestations of 76 episodes of hospital-acquired (HA) and 28 episodes of community-acquired (CA) rotavirus infection in neonatal care units[a]

	Total	HA group ($n = 76$)	CA group ($n = 28$)	p value
Demographics				
Postnatal age at onset of illness (d)	29 ± 27 (1～131)	24.7 ± 2.8	33.9 ± 6.4	0.29
Postmenstrual age at onset of illness(wk)	37.6 ± 5 (26.7～52.7)	36.1 ± 0.5	41.8 ± 0.6	<0.001
Birth weight (g)	2137 ± 953 (460～4800)*	1793 ± 101	2872 ± 138	<0.001
Gestational age (GA) (wk)	33.7 ± 4.5 (24～40)*	32.14 ± 0.49	36.96 ± 0.67	<0.001
GA ≥ 37 wk	34 (34%)	14 (18.4%)	20 (71.4%)	<0.001
34 wk ≤ GA < 37 wk	20 (20%)	14 (31.6%)	6 (21.4%)	0.42
GA < 34 wk	46 (46%)	48 (50.0%)	2 (7.1%)	<0.001
Body weight at onset (g)	2650 ± 1249 (480～5888)	2229 ± 125	3779 ± 173	<0.001
Male gender	56 (56%)*	42 (55.3%)	16 (57.1%)	0.86
Underlying diseases	65 (65%)*	57 (75.0%)	8 (28.6%)	<0.001
Hospitalization days of onset in HA group	–	23 ± 25 (−3～131)	–	–
Clinical manifestations*				
Poor activity and appetite	24 (23.1%)	14 (18.4%)	10 (35.8%)	0.06
Fever	21 (20.2%)	9 (11.8%)	12 (78.6%)	<0.001
<12 h	13 (61.9%)	6 (66.7%)	7 (58.3%)	–
12–24 h	4 (19.1%)	1 (11.1%)	3 (25.0%)	–
24–48 h	4 (19.1%)	2 (22.2%)	2 (8.3%)	–
>48 h	0	0	0	–
Vomiting	22 (21.2%)	13 (17.1%)	9 (32.1%)	0.10
Stool character				
Watery	14 (13.5%)	7 (9.2%)	8 (28.6%)	0.04
Loose	55 (52.9%)	36 (47.3%)	19 (67.9%)	0.06
Blood or mucus	44 (42.3%)	40 (52.6%)	4 (14.3%)	<0.001
Constipation	10 (9.5%)	6 (7.9%)	4 (14.3%)	0.33
Abdominal distension	54 (51.9%)	40 (52.6%)	14 (50.0%)	0.81
Feeding intolerance	16 (15.4%)	12 (15.8%)	3 (10.7%)	0.51
Unstable vital signs	34 (32.7%)	30 (39.5%)	4 (14.3%)	0.02
Body weight loss	1 (1.0%)	0	1 (3.6%)	
Seizure	1 (1.0%)	0	1 (3.6%)	
Conscious disturbance	1 (1.0%)	0	1 (3.6%)	
Bulging fontanelle	0	0	0	
Others[b]	1 (1.0%)	1	0	
Complications (%)				
Dehydration	4 (3.8%)	3 (3.9%)	1 (3.6%)	0.93
Metabolic acidosis	22 (21.2%)	18 (23.7%)	4 (14.3%)	0.30
Respiratory failure	3 (2.9%)	3 (3.9%)	0	0.28
Urinary tract infection	2 (1.9%)	1 (1.3%)	1 (3.6%)	0.45
Co-infection[c]	12 (11.5%)	8 (10.5%)	4 (14.2%)	0.59
Sepsis	9 (8.7%)	9 (11.8%)	0	0.06
Necrotizing enterocolitis (NEC)	22 (21.2%)	20 (26.3%)	2 (7.1%)	0.03
Stage I	10[d]	9	1	
Stage II	10[e]	8	1	
stage III	2[f]	3	0	
Coagulopathy	3 (2.9%)	3 (3.9%)	0	0.29
Ileus requiring rectal tube for decompression	3 (2.9%)	1 (1.3%)	2 (7.1%)	0.12
Death	5 (4.8%)	4 (5.3%)	1 (3.6%)	0.72
Recurrence	4 (3.8%)			

* The demographic data and recurrence were counted based on 100 patients, and the clinical manifestations and complication were counted based on 104 episodes.

[a] Data presented as median ± SD (range) or n (%).

[b] The other manifestation was upper gastrointestinal tract bleeding.

[c] The sites of co-infection included two pneumonia, nine bacteremia, two urinary tract infection, one salmonellosis, one norovirus infection and one respiratory syncytial virus infection.

[d] Of the 10 episodes of NEC stage I, two belonged to Ia, and eight belonged to Ib.

[e] Of the 10 episodes of NEC stage II, eight belonged to IIa, and two belonged to IIb.

[f] The two episodes of NEC stage III were both stage IIIb.

Table 2 Comparisons of laboratory findings of the infants with 76 episodes of hospital-associated (HA) and 28 episodes of community-associated (CA) rotavirus Infection

	Total	HA group	CA group	*p* value
WBC ($\times 10^3/mm^3$) (93*)	10.5 ± 4.30 (2.2～23.3)	10.1 ± 0.54	11.54 ± 0.77	0.15
Leukocytosis[a]	4 (4.3%)	4.6%	3.6%	—
Leukopenia ($<5{,}000/mm^3$)	7 (7.5%)	9.2%	3.6%	—
Haemoglobin (g/dL) (93*)	12.3 ± 2.3 (8.2～19.3)	12.2 ± 2.1	12.5 ± 2.8	0.68
Platelets ($\times 10^3/L$) (93*)	309 ± 150 (9～842)	279.3 ± 18.0	380.8 ± 26.6	0.002
Thrombocytosis ($>450 \times 10^3/L$)	25 (26.9%)	21.5%	39.3%	0.07
Thrombocytopenia ($<150 \times 10^3/L$)	14 (15.1%)	20%	3.6%	0.04
Creatinine (54*)	0.45 ± 0.41 (0.1～2.8)	0.53 ± 0.08	0.31 ± 0.02	0.05
AST (mg/dL) (53*)	43.5 ± 47.2 (6～327)	39.3 ± 5.3	50.0 ± 14.4	0.51
ALT (mg/dL) (38*)	23.7 ± 16.3 (2.6～6.9)	20.4 ± 2.9	26.2 ± 4.1	0.36
Sugar (mg/dL) (46*)	113.3 ± 66.7 (57～362)	120.1 ± 13.2	103.3 ± 15.3	0.41
Potassium (mEq/L) (80*)	4.8 ± 0.8 (2.6～6.9)	4.7 ± 0.8	5.3 ± 0.7	0.002
Hyperkalemia (>5.0 mEq/L)	32 (40%)	30.9%	60.0%	0.04
Na (mEq/L) (80*)	136.8 ± 3.5 (126～147)	136.9 ± 3.9	136.6 ± 2.7	0.69
Hyponatremia(<135 mEq/L)	15 (18.8%)	20%	16%	0.71
Chloride (mEq/L) (72*)	106.2 ± 6.03(91～130)	105.9 ± 6.7	107.2 ± 3.9	0.40
C-reactive protein (mg/L) (91*)	16.76 ± 32.57 (0.2～164)	18.1 ± 4.3	13.5 ± 5.3	0.54
>40 (mg/L)	12(13.2%)	14.1%	11.1%	0.70
pH (56*)	7.35 ± 0.12 (6.8～7.6)	7.350 ± 0.12	7.40 ± 0.08	0.35
Base deficit (mEq/L) (56*)	4.2 ± 7.7 (-14.8～28)	3.4 ± 1.1	10.3 ± 3.0	0.04
Stool occult blood (91*)	62 (68.1%)	69%	65%	0.72
Pus on stool routine (90*)	14 (15.6%)	15.6%	15.4%	0.97
Mucus on stool routine (90*)	22 (24.4%)	15 (23.4%)	7 (26.9%)	0.73
Salmonella on stool culture (74*)	1 (1.4%)	0	4.8%	0.23
Campylobacter on stool culture (73*)	0	0	0	—

* The number of samples (*n*).
[a] Leukocytosis was defined as a leukocyte count $\geq 34{,}000/mm^3$ for patients aged within 1 week; $\geq 19{,}500/mm^3$ for patients aged between 1 week and 1 month; and $\geq 17{,}500/mm^3$ for patients aged between 1 month and 1 year.
ALT = alanine transaminase; AST = aspartate aminotransferase; WBC = white blood cell.

bronchopulmonary dysplasia, who developed a second episode of RV infection at the age of 81 days. Ventilator-associated pneumonia caused by *Enterobacter cloacae* was noted simultaneously and the infant died 14 days later. The second case was a full-term infant, subsequently diagnosed with spinal muscular atrophy, who presented to our emergency department as an out-of -hospital cardiac arrest at the age of 52 days. Blood and mucus in the stool was noted on admission and subsequently proved to be RV infection. He died of severe hypoxic-ischemic encephalopathy 7 days later. The third case presented with bradycardia, cyanosis and severe metabolic acidosis within 3 days of discharge from our hospital. Blood in the stool was noted on the second hospital day, and was documented to be RV infection. The infant died on hospital day 3 due to profound metabolic acidosis, and was subsequently proved to be inborn error metabolic disease. The fourth case, a premature infant with a GA of 24 weeks and underlying diseases of pulmonary hypertension and bronchopulmonary dysplasia, developed sepsis and bloody, mucoid stool at 31 days of age. Abdominal distension, and massive ascites followed later. The infant died of deteriorated gastrointestinal symptoms with septic shock and respiratory failure 6 days later. Negative bacterial culture for urine, blood, stool and sputum was found later, but the stool specimen was positive for RV antigen. The fifth case presented with feeding intolerance, abdominal distension and poor activity at 8 days old in a regional nursery setting and was transferred to our hospital. Complex congenital heart disease with acute heart failure was diagnosed thereafter. The infant died on the next day after extracorporeal membrane oxygen was administered for profound metabolic acidosis at the age of 11 days.

Discussion

Results from the present study showed that three-quarters of the infants hospitalized in neonatal care units with rotaviral infections were HA cases and more than 60% of these were preterm infants. These findings were consistent with those reported previously by Dearlove et al, i.e., that

Table 3 Comparisons of clinical manifestations of young infants, stratified by gestational ages, with hospital-acquired rotavirus infection

Gestational age	≥37 wk	34～<37 wk	<34 wk
	(n = 14)	(n = 14)	(n = 48)
Clinical manifestations			
Poor activity and appetite	2 (14.3%)	1 (7.1%)	11 (22.9%)
Fever**	6 (42.9%)	0	3 (6.3%)
Stool character			
Blood or mucus in stool*	6 (42.9%)	6 (42.9%)	29 (60.4%)
Watery stool*	4 (28.6%)	2 (14.3%)	1 (2.1%)
Vomiting	2 (14.3%)	1 (7.1%)	10 (20.8%)
Feeding intolerance	0	3 (21.4%)	9 (18.8%)
Constipation	2 (14.3%)	2 (14.3%)	2 (4.2%)
Unstable vital signs*	3 (21.4%)	2 (14.3%)	25 (52%)
Abdominal distension	5 (35.7%)	5 (35.7%)	30 (62.5%)
Complication			
Necrotizing enterocolitis**	0	1 (7.1%)	19 (39.6%)
Sepsis	2 (14.3%)	1 (7.1%)	6 (12.5%)
Dehydration	2 (14.3%)	1 (7.1%)	10 (20.8%)
Metabolic acidosis	4 (28.6%)	3 (21.4%)	11 (22.9%)
Death	1 (7.1%)	1 (7.1%)	2 (4.2%)

*Significant difference ($p < 0.05$).
**Significant difference ($p < 0.01$).

smaller and sicker babies who stay in hospital longer are more likely to acquire RV infections.[11] The present study also indicated that the clinical manifestations of RV gastroenteritis in this age group were significantly different between those with CA and HA infections, as well as between term and preterm infants. Generally, most CA cases were term babies; term babies more often presented with fever and watery stools, which is a picture similar to that seen in elder infants and children; while preterm babies more often presented with bloody, mucoid stool and NEC, which is a picture different from that seen in elder infants and children. These findings were consistent with those of previous studies.[11–13] In addition, the infants with CA infections in the present study had a higher rate of hyperkalemia, and base deficit, indicative of dehydration.

Some studies have shown a higher proportion of advanced NEC in term infants with RV infections,[12] while others have reported that NEC is more common in smaller-weight infants.[11–13] In the present study, NEC was more commonly seen among preterm neonates, and nearly 40% of preterm neonates in the HA group presented with NEC. Although rotaviruses can be implicated in NEC,[14–16] the issue whether there is a causal effect between rotaviral infection and NEC should be further studied. However, since we observed that those premature infants with necrotizing enterocolitis had RV infection simultaenously in the current study, we suggest that if a neonate, especially a preterm infant, presents with NEC, RV infection should be included in the differential diagnoses and contact precautions should be implemented strictly until proved otherwise.

Previous studies hypothesized that the different clinical presentations of RV infections in this age population may be linked to maturational changes in the neonatal intestine.[17,18] During the neonatal period, the gastrointestinal (GI) tract alters its absorptive and secretary functions to accommodate changes in the availability of substrates, which occur more slowly in the immature intestine.[17] These factors combine to develop GI tract dilatation and ischemia in a preterm host, instead of secretion and increasing peristalsis. However, whether RV infection is a risk factor or an exacerbating factor of NEC remained controversial.[16] Although RV infection was not the only cause of the five deaths in our study, it is true that the infants' GI symptoms had deteriorated their underlying conditions. Therefore, RV was sometimes fatal, especially for those critically ill neonates.

There are several limitations in this study, since it is a retrospective study in nature. First, since RV antigen immunoassay was performed in the infants only when clinically suspected, not every patient with RV infection, if not clinically suspected, was included in this study. Therefore, the clinical features shown here cannot represent the whole picture of neonates and young infants with RV infections. Secondly, testing for other potential pathogens as well as RV antigen was not performed in each case, although stool cultures for bacteria were performed in 74 episodes (71%) with one positive for *Salmonella*. Thus, we could not exclude the possibility of co-infection. Additionally, we did not survey the effect of breastfeeding on RV infection, which has been reported to be a protective factor for RV infection,[11,12,19] since this information was difficult to obtain from the medical records retrospectively.

In summary, RV infections among neonates and young infants generally do not present with watery stools, diarrhea or dehydration. In contrast, abdominal distention and bloody mucoid stools are commonly associated with RV infections in neonatal care settings. Preterm infants more commonly present with bloody mucoid stools, while fever are more common in term infants. A substantial proportion of RV infections in neonatal care units may develop NEC, especially in preterm neonates. Once introduced, RV appears to become a troublesome problem of HA infections in neonatal care settings. The high ratio of HA RV infections in neonatal care units emphasizes the need for routine strict infection control measures in this setting.

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