ÇOCUKLARDA VİRAL GASTROENTERİTLER

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Gazi Üniversitesi Tıp Fakültesi
Çocuk Sağlığı ve Hastalıkları ABD
Çocuk Enfeksiyon Bilim Dalı

21. Yüzyılda ishal

- Her yıl dünyada yaklaşık 2 milyar ishal oluyor
- Genellikle 2 yaşın altındaki çocuklar etkileniyor
- İshalli hastalıklara bağlı yılda 1.5 milyon çocuk ölümü
- Dünyada enfeksiyonlara nedenli ölümler içerisinde ilk iki sırada
- Beş yaşın altındaki çocuklarda malnutrisyon sebebi

http://www.who.int/mediacentre/factsheets/fs330/en/index.html

21. Yüzyılda ishal ABD'de

- Yılda yaklaşık 2-3 milyon poliklinik viziti
- Yatışların %10'u ishal
- 200.000 hastaneye yatış
- Her yıl yaklaşık 300 ölüm

King CK, Glass R, Bresee JS, Duggan C; Centers for Disease Control and Prevention. Managing acute gastroenteritis among children: oral rehydration, maintenance, and nutritional therapy. MMWR Recomm Rep. 2003;52(RR-16):1–16.

Gastroenteritler

- Gelişmekte olan ülkelerde surveryans zor
- Laboratuar tekniklerinin gelişmesi, bazı mikroorganizmaların patojen olarak önem kazanmasında rol oynamaktadır
- Teknolojik ilerleme, enfeksiyöz ishal etkenlerinin virülans faktörlerinin, hastalığın fizyopatolojisinin anlaşılmasında ve hastalığın tedavisinde yeni gelişmeler sağlamıştır

Çocuklarda akut ishal nedenleri

- Viruses
  - Rotaviruses
  - Noroviruses (Norwalk-like viruses)
  - Enteric adenoviruses
  - Caliciviruses
  - Astroviruses
  - Enteroviruses
- Bacteria
  - *Campylobacter jejuni*
  - Nontyphoid *Salmonella* spp
  - Enteropathogenic *Escherichia coli*
  - *Shigella* spp
  - *Yersinia enterocolitica*
  - Shiga toxin producing *E coli*
  - *Salmonella typhi* and *S paratyphi*
  - *Vibrio cholerae*
- Protozoa
  - *Cryptosporidium*
  - *Giardia lamblia*
  - *Entamoeba histolytica*
- Helminths
  - *Strongyloides stercoralis*

Viral etkenler

- Rotavirus (en sık)
- İnsan calisivirus
- Adenovirus
- Astrovirus
- Diğer
- Parvovirus B19
- Enterovirus
- Coronavirus
- Torovirus
- Picobirnovirus
- Bocavirus
- CMV

•Benzer bulaşma özellikleri
•Klinikleri benzer
•Tedavileri benzer
•Çoğunda tanı konulamaz

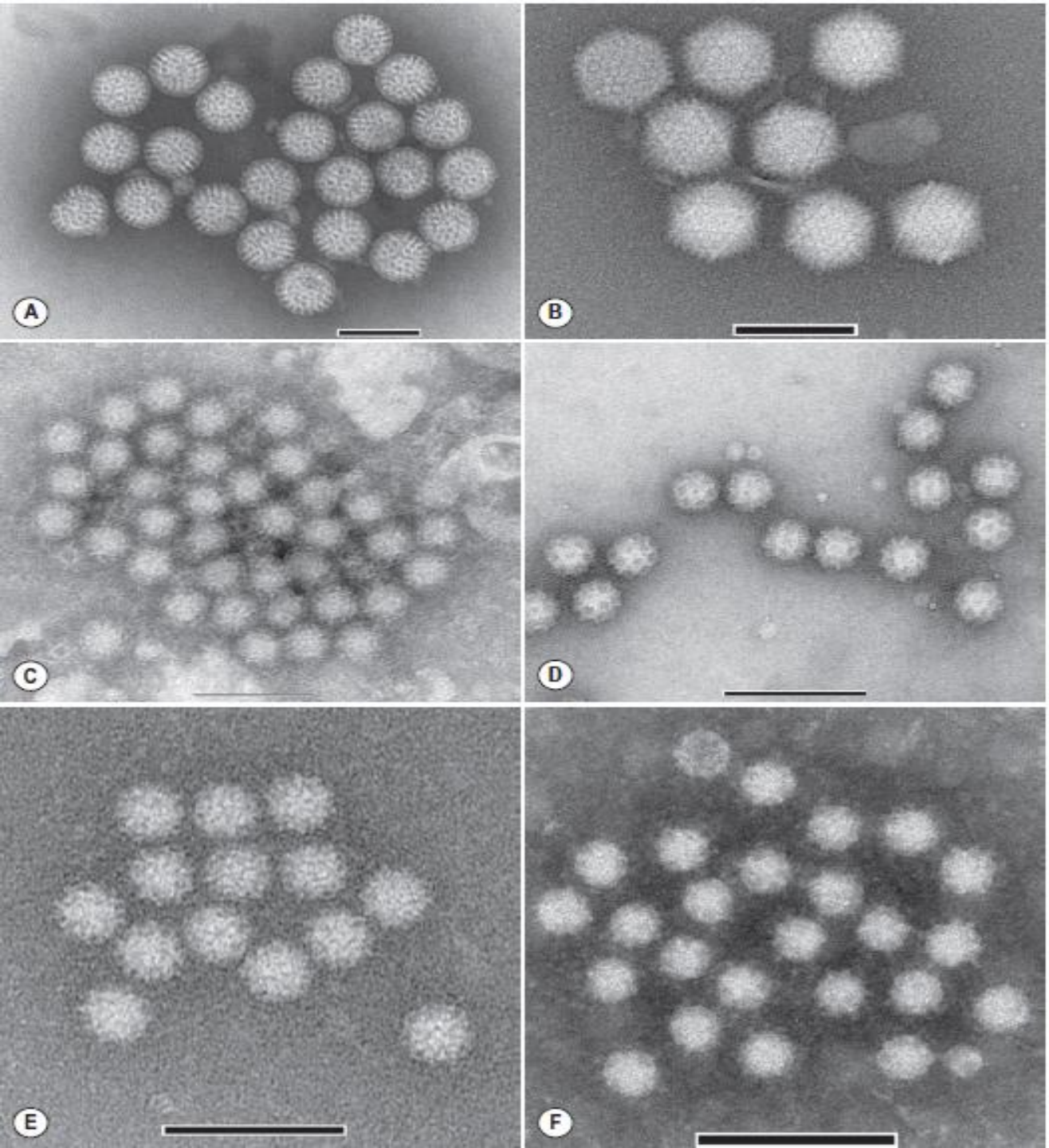


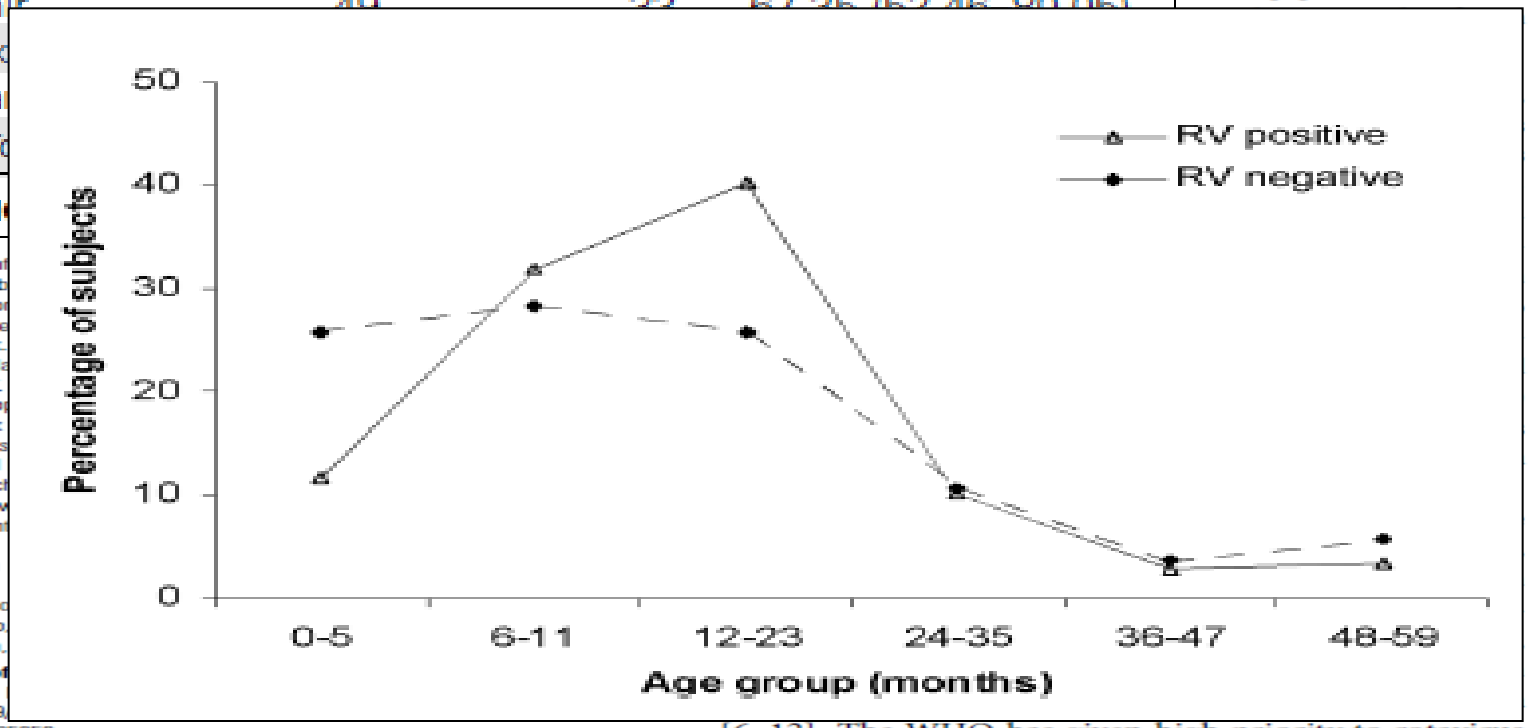
Figure 58-1. Electron micrographs of four viruses that are known to cause gastroenteritis. **(A)** Rotaviruses are 70- to 80-nm multi-shelled particles; the inner shell has a visible "wheel and spoke" character. **(B)** Adenoviruses are 70- to 90-nm icosahedral structured viruses with fiber extensions on their vertices. The fibers are fragile and not always seen on cell culture prepared adenovirus or on virus seen in fecal suspensions. **(C–E)** Noroviruses and sapoviruses are 33- to 40-nm viruses. Fecal suspension particles often are coated with gastrointestinal derived antibodies as shown in panel **C.** Human noroviruses and sapoviruses are fastidious but virus-like particles (VLPs) formed of capsid proteins can be produced in recombinant-based cultures, as shown in panels **D** and **E.** The calicivirus VLPs may be slightly larger (37 to 41 nm) than their respective viruses but have typical calicivirus structure. Panel **D** VLPs were derived from sapovirus and panel **E** from norovirus. The "star of David" image associated with caliciviruses is readily apparent on sapovirus VLPs **D** but is not visible on norovirus VLPs **E. (F)** Astroviruses are 25–30 nm, have a smooth edge, and a distinctive 5- or 6-pointed star on some particles in fecal-suspension derived virus. The smooth surface is not always present on astroviruses grown in culture and can resemble miniature versions of noroviruses. Scale bars = 100 nm. (Courtesy of Charles D. Humphrey, PhD, CDC, Atlanta, GA.)

Multicenter Prospective Study on the Burden of Rotavirus Gastroenteritis in Turkey, 2005–2006: A Hospital-Based Study

Table 1. Proportion of Rotavirus Gastroenter… Children Hospitalized for Gastroenteritis … Rotavirus

Center	Total no. of children hospitalized for gastroenteritis and tested for rotavirus	No.	Percentage (95% CI)
Adana	105	34	32.38 (23.57–42.21)
Izmir	49	33	67.35 (52.46–80.05)
Ank…			
Ista…			
To…			

Rotavirüs %53



[6–12]. The WHO has given high priority to rotavirus

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Geliş Tarihi / *Received*: 16.07.2010 • Kabul Tarihi / *Accepted*: 24.11.2010

Tablo 2. Etken saptanan hastaların yaş gruplarına göre dağılımı

	0-6 ay	7-12 ay	13-24 ay	25-60 ay	>60 ay	Toplam	p
Rotavirüs	23(%12.1)	59 (%31.1)	53(%27.9)	35(%18.4)	20 (%10.5)	190 (%64.6)	>0.05
Adenovirüs	4 (%33.3)	0 (%0)	3 (%25)	4 (%33.3)	1 (%8.3)	12 (%4.1)	-*
Rotavirüs + Adenovirüs	2 (%16.7)	3 (%25)					
Amibiyazis	4 (%5.3)	15 (%					
Salmonella	0(%0)	0 (%					
Shigella	1(%50)	0(%0)					-*
Giardiazis	0 (%0)	0 (%0)	0 (%0)	0 (%0)		1(%0.35)	-*
Toplam	34 (%11.6)	77 (%26.2)	67 (%22.8)	63 (%21.4)	53 (%18.0)	294 (%100)	

•% 57.7 hastada etken saptandı
•% 37.3 rotavirus

* Vaka sayısı yetersizliği nedeniyle istatistiksel bilgi verilememiştir

ÖZET

Akut gastroenterit çocukluk çağında sık görülen öldürücü hastalıklar arasında ilk sırada yer almaktadır. Bölgedeki gastroenterit etkenlerinin bilinmesi, tanıya yönelik araştırmalarda kolaylık, erken doğru tanı ve etkin tedavi fırsatı sağlayacak, ayrıca antimikrobiyal tedavi gereken durumlarda antibiyotik seçimi için yol gösterecektir. Bu çalışmanın amacı, Ocak 2008-Ocak 2009 tarihleri arasında Ankara'da bir referans hastanesi olan hastanemize akut gastroenterit nedeniyle başvuran 509 olguyu epidemiyolojik ve klinik bulgular yönünden değerlendirmektir. Gaita örnekleri bakteriyel, viral ve parazitik enteropatojenler açısında mikroskopik inceleme, immünolojik testler ve bakteriyel kültür methodları kullanılarak değerlendirilmiştir. Olgularımız bir yıl boyunca çeşitli nedenlerle hastaneye yatan hastaların %9.3'ünü oluşturmuştur. Hastaların %57.7'sinde (n:294) ishale neden olan etken saptanırken, %42.3'ünde etken saptanamadı. İshalli vakaların büyük çoğunluğunu erkekler oluşturuyordu. Rotavirüs en sık ilk 2 yaşta, amibiyazis ise en sık 5 yaş üzerinde tespit edildi. Rotavirüs en sık Nisan ayında (%16.8) ve ilkbahar mevsiminde (%43.7) görüldü. Amibiyazisli olgular en sık Eylül ayında (%14.5) ve yaz mevsiminde tespit edildi (%32.9). Viral etkenlere bağlı hastanede yatış süreleri daha uzun bulunurken (rotavirüs 4 gün, adenovirüs 5.7 gün, rotavirüs+adenovirüs 4.5 gün), amibiyazisli olgularda daha kısa (3 gün) bulundu.

Anahtar Sözcükler: Akut gastroenterit, çocuklar, hastaneye yatış

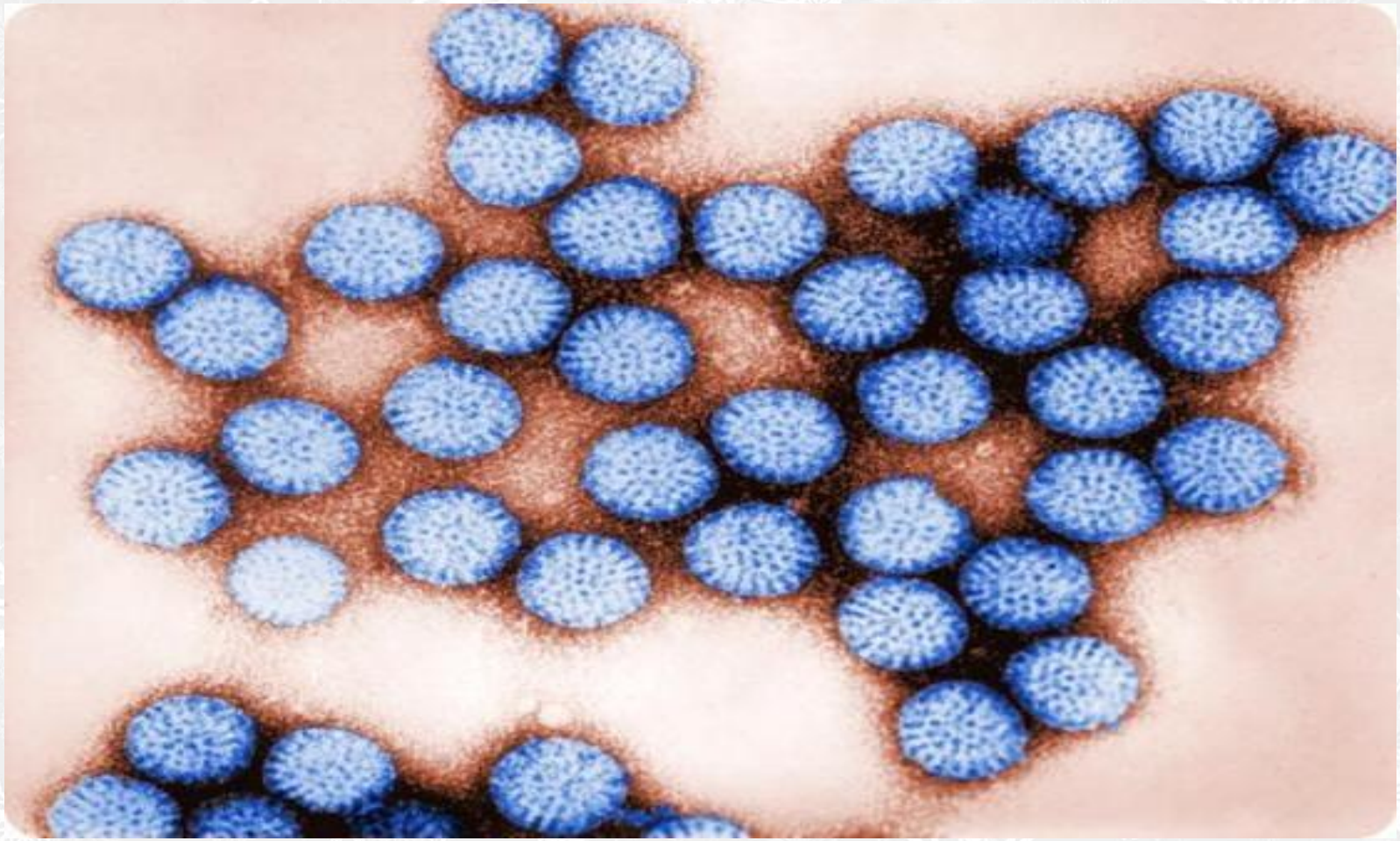
Epidemiyoloji

- Endemik hastalık
- Epidemik hastalık

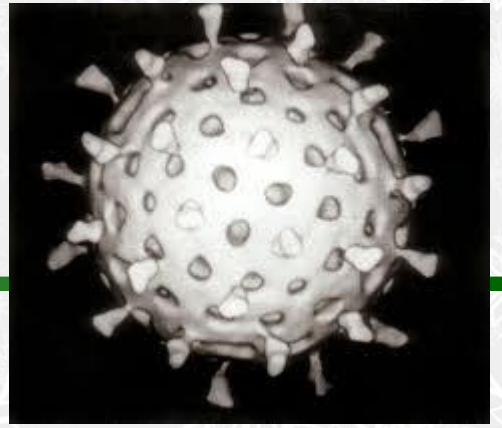
- **Rotavirus, astrovirus, enterik adenovirus,** ve **sapovirus** primer olarak **endemik** hastalıkken, norovirus infeksiyonları hem endemik hastalık hemde salgınlarla seyreder
- Sık görülen etkenler coğrafik bir özellik göstermez
- Ilıman ülkelerde rotavirus, astrovirus ve sapovirus kışın görülürken, tropikal bölgelerde bütün yıl görülebilir
- Norovirus bütün mevsimlerde sirküle olur, ancak salgınlar daha çok kışın görülür

Charles MD. Pediatr Infect Dis J 2006;25:489–493.
Cook SM. Bull World Health Organ 1990;68:171–177.
Lopman BA. Emerg Infect Dis 2003;9:71–77.
Lebaron CW. Pediatr Infect Dis J 1992;11:860–865.

Rotavirus



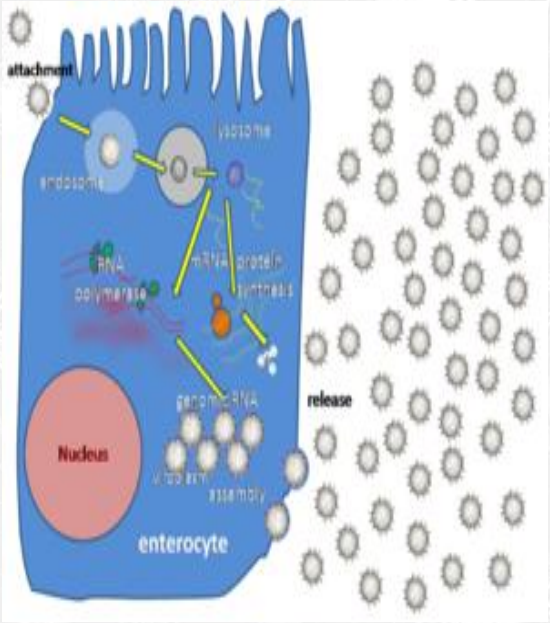
Rotavirus



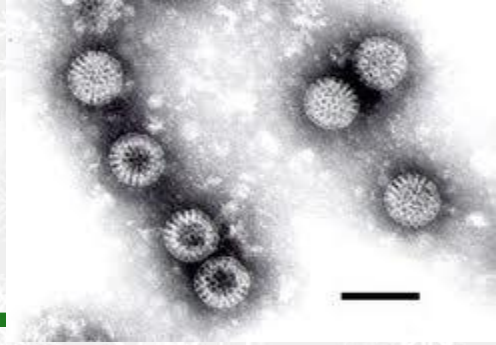
- Rotavirüsler, reovirüs ailesinde yer alıp, 11 segmentli, çift zincirli RNA taşırlar

- Yapısal 6 protein (VP1-VP4, VP6 ve VP7) ve yapısal olmayan 6 proteini (NSP1-NSP6) kodlar

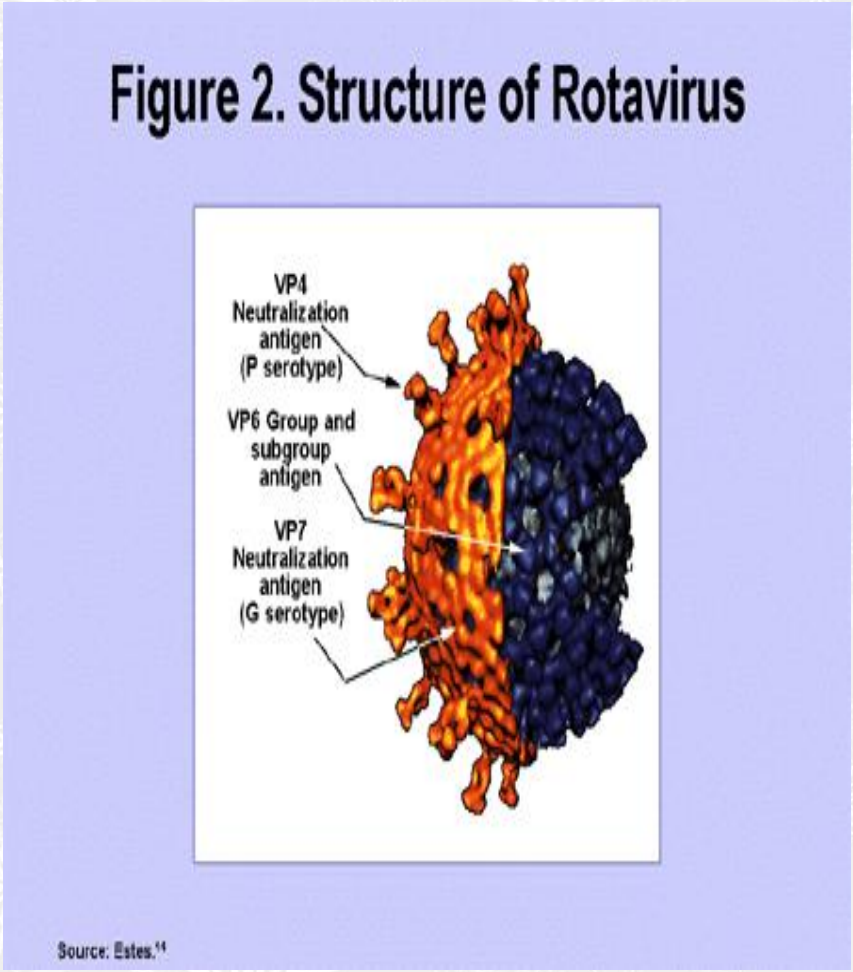
- Zarfları bulunmaz, sitoplazma içerisinde çoğalırlar



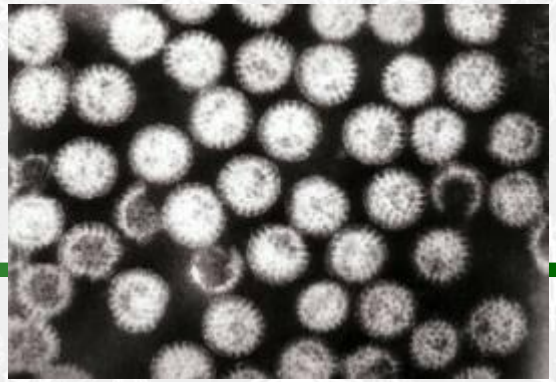
Rotavirus



- Dış kapsit, iç kapsit ve çekirdek şeklinde üç katmandan oluşur
- Dış kapsit yapısal 2 proteinden meydana gelmiştir (P protein olarak adlandırılan VP4 ve G protein olarak bilinen VP7)
- VP4 ve VP7 doğal enfeksiyon sırasında nötralizan antikor cevabını uyarır, serotip belirlenmesinde kullanılır
- 14 G serotipi ve 20 P serotipi var
- İç kapsid, VP6 proteininden meydana gelmiştir.
- Bu protein virionun en çok bulunan ve en immunojenik olan proteinidir



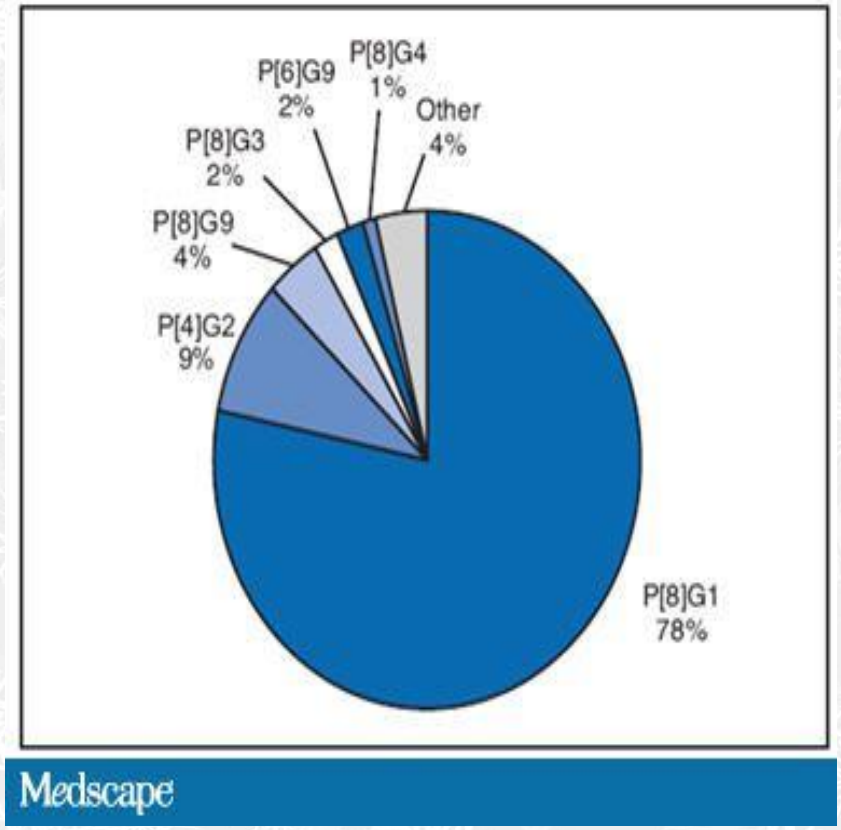
Rotavirus



- Rotaviruslar 7 serogrup ve serotipe ayrılmıştır
- Farklı 7 serogrup var (A-G) (VP6'daki farklılığa göre)
- Sadece A, B ve C grupları insanları enfekte eder
- İnsanlarda en sık görülen A grubu
- Serotiplendirme sadece A grubu için yapılmıştır

Rotavirus

- Endüstrileşmiş ülkelerde %90'ın üzerinde, Güney Amerika ve Asya ülkelerinde %68 oranında etken G serotip, G1, G4, G9 ve P genotip, P4 ile P8'dir.
- Tüm dünyada hakim olan G1P[8] olup, bunu G3P[8], G2P[4], G4P[8] ve G9[P8] takip eder.
- Bu beş rotavirüs suşu dünya genelinde görülen suşların %50-90'sını oluşturur



Steele AD. RotaVaccine 2003; 21: 361-367.

Multicenter Prospective Study on the Burden of Rotavirus Gastroenteritis in Turkey, 2005–2006: A Hospital-Based Study

Mehmet Ceyhan,[1] Emre Alhan,[2] Nuran Salman,[3] Zafer Kurugol,[4] Inci [illegible] Guldane Koturoglu,[4] Hasan Tezer,[1] Emine Kuset Bulbul,[2] Metin Ka[illegible] Robert Pawinski[6]

[1]Department of Pediatrics, Faculty of Medicine, Hacettepe University, Ankara, [2][illegible] Adana, [3]Department of Pediatrics, Faculty of Medicine, Istanbul University, Istanb[illegible] University, Izmir, Turkey; [5]GlaxoSmithKline Biologicals, United Arab Emirates; and

•G1P8 %76 en sık

•Sonra G2P4, G9P8

Rotavirus is the main cause of gastroenteritis and dehydration req[illegible] [illegible]nts and children. Despite the high diarrhea-related mortality rate, there are limited stud[illegible]ng the prevalence of rotavirus in Turkey. The disease burden of rotavirus ga[illegible]enteritis in Turkey was assessed by active, prospective surveillance conducted in accordance with a modified World Health Organization generic protocol from 1 June 2005 through 1 June 2006. A total of [illegible] children aged <5 years who were hospitalized for gastroenteritis in 4 centers were enrolled. Rotavirus was identified in 53% of samples from the 338 children tested; the range for individual centers was 32.4%–67.4%. Overall, 83.8% of rotavirus-positive children were aged <2 years. Rotavirus gastroenteritis occurred year-round but peaked in the winter. G1P[8] was the most widely prevalent strain (76% of strains), followed by G2P[4] (12.8%). G9P[8] was reported in samples from 3.9% of children. These data support the need for a rotavirus vaccine in Turkey.

Acute diarrhea in early childhood is among the most important challenges in public health [1] and is considered by the World Health Organization (WHO) to be one of the leading causes of childhood mortality [2].

Among all enteric pathogens that infect young children, rotavirus is the primary cause of severe gastroenteritis worldwide, affecting nearly all children by age 5 years and accounting for ~39% (range, 29%–45%) of all diarrhea-related hospitalizations [3]. Although the incidence of rotavirus is similar in developing and developed countries, the mortality rate is higher in developing countries, likely because of poor access to hydration therapy and a high prevalence of malnutrition [1]. Global mortality due to rotavirus is estimated to be 527,000 deaths (range, 475,000–580,000 deaths) among children aged <5 years, with >80% of the deaths occurring in the developing countries of South Asia and sub-Saharan Africa [4,5].

Two vaccines (RotaTeq [Merck], licensed in the United States, Europe, and >69 countries worldwide, and Rotarix [GlaxoSmithKline Biologicals], licensed in the United States, Europe, and >100 countries worldwide) have demonstrated good safety, immunological, and efficacy profiles in large clinical trials conducted in western industrialized countries and in Latin America [6–12]. The WHO has given high priority to rotavirus

Potential conflicts of interest: M.C., E.A., N.S., Z.K., I.Y., U.C., M. Keser, G.K., E.K.B., M. Karabocuoglu, and O.H. received honoraria or paid expert testimony or travel grants from GlaxoSmithKline Biologicals. M. Keser or M. Karabocuoglu also have personal relationships with other people or organizations that could bias the submitted work. S.A. is an employee of GlaxoSmithKline Biologicals. R.P. is an employee of GlaxoSmithKline Biologicals and a shareholder in GlaxoSmithKline Biologicals. H.T. reports no potential conflicts.

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Reprints or correspondence: Prof. Mehmet Ceyhan, Pediatrics Dept., Hacettepe Universitesi Tip, Fakultesi Çocuk Sagligi ve Hastaliklari Anabilim Dali-06100 Sihhiye, Ankara, Turkey (mceyhan@hacettepe.edu.tr).

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DOI: 10.1086/605056

Curr Microbiol (2011) 63:517–522
DOI 10.1007/s00284-011-0014-2

Changing Patterns of Rotavirus Genotypes i

Anil Tapisiz · Zeynep Ceren Karahan ·
Ergin Çiftçi · Erdal İnce · Ülker Doğru

•494 dışkı örneği

•%28.1 rotavirus

•%55'inde G9P8

Received: 17 June 2011 / Accepted: 12 September 2011 / Published online: 22 September

Abstract To describe the circulation dynamics of human rotavirus genotypes and to understand the epidemiological changes of rotavirus infection in Turkey, one of the countries where the highest mortality rates are seen due to rotavirus in Europe. Stool samples of children under 5 years of age which gave positive results for rotavirus antigen were stored at −20°C and then genotyped using multiplex reverse transcription polymerase-chain reaction. Of the 494 stool samples, 137 (28.1%) were positive for rotavirus antigen and 100 (73%) samples which could be genotyped successfully were included in the study. 42 (42%) samples were from inpatients, and 58 (58%) were from outpatients. The median age of the children was 16 months (5 days–59 months). G9 and P[8] were the most frequent G and P genotypes, and were detected in 30 (30%) and 55 patients (55%), respectively. In 90 samples for which both G and P genotypes could be determined, 34 different combinations were found. G9P[8] was the most frequent genotype detected in 19 patients (19%), followed by G1P[8] and G4P[6] each in 7 (7%) patients. The incidence of mixed infection was found to be 26%. Novel strains like P2A[6] and P[5] and unusual reassortant strains were detected. Distribution of rotavirus genotypes exhibited distinctive changes in this study. When the ever-changing epidemiology of rotaviruses is taken into account, ongoing surveillance studies are important before the inclusion of rotavirus vaccines in national immunization program of Turkey.

Introduction

Rotavirus is the most common cause of severe gastroenteritis in children under 5 years of age [29]. Worldwide it causes an estimated 600,000 deaths and more than 2 million hospitalizations each year [10, 30]. Group A, B, and C rotaviruses are associated with human disease but globally group A rotaviruses are the major cause of childhood diarrhea [3]. Group A rotaviruses have also been identified in a wide variety of animal species worldwide. The rotavirus genome is consisted of 11 segments of double-stranded (ds) RNA, which encode six structural proteins (VP1-4, VP6-7) and five non-structural proteins (NSP1-5). Serotype specificity is determined by the outer capsid proteins VP4 (P) and VP7 (G) [17]. To date ,27 G and 35 P genotypes have been determined. The G1–G4 and G9, and P[4], P[6], and P[8] genotypes are the most common causes of rotavirus disease in children [9, 28]. However, rotavirus strains with other G and P types have increasingly been reported in different parts of the world [27, 28]. Surveillance studies monitoring the genotype changes are important because of the segmented nature of rotaviruses.

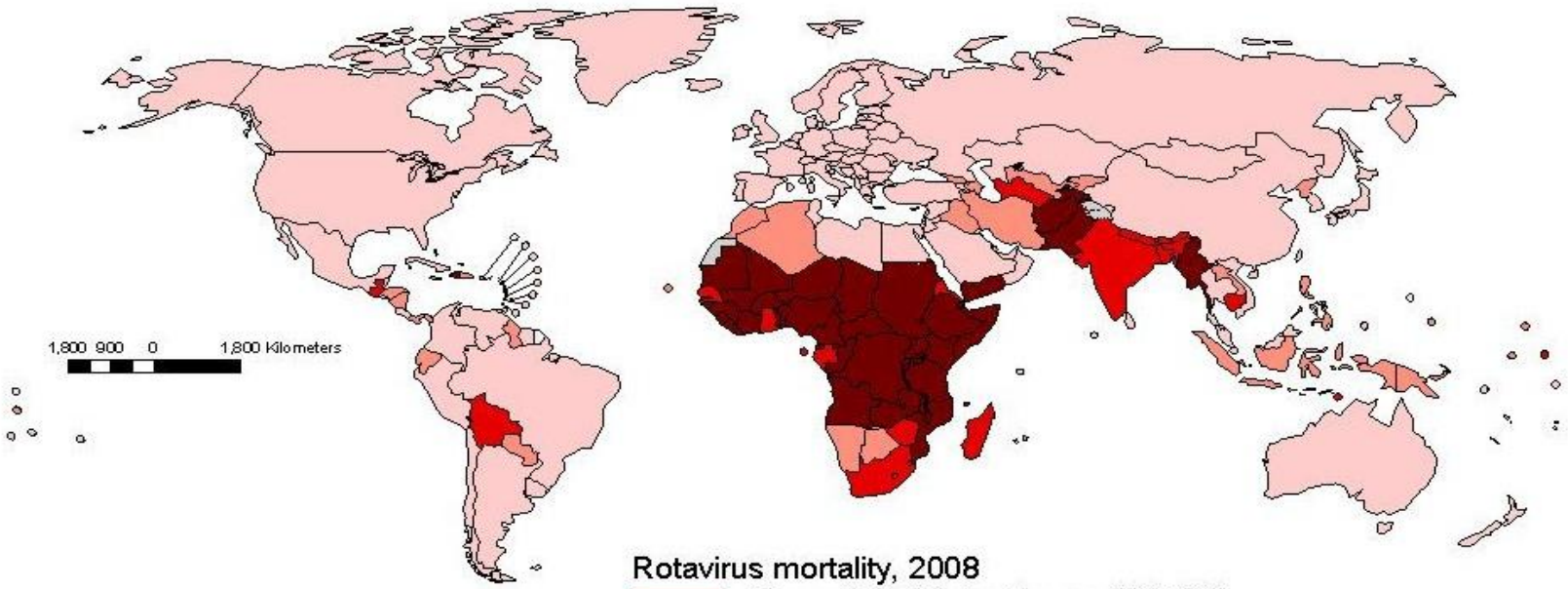
Rotavirus

- Rotavirus ishali nedeniyle dünya genelinde her yıl yaklaşık 25 milyon poliklinik başvurusu
- Sıklıkla 2 yaşın altında görülür
- Beş yaşına gelen bir çocuk, en az 1 kez rotavirus enfeksiyonu geçirmiştir
- Her yıl 2 milyon çocuk hastaneye yatırılmakta 600.000'den fazla çocuk kaybedilmektedir.
- İshal ilişkili hospitalizasyonların %39 nedeni

Parashar UD. Emerg Infect Dis 2003;9: 565–72.

Rotavirus

Rotavirus mortality in children younger than 5 years, 2008



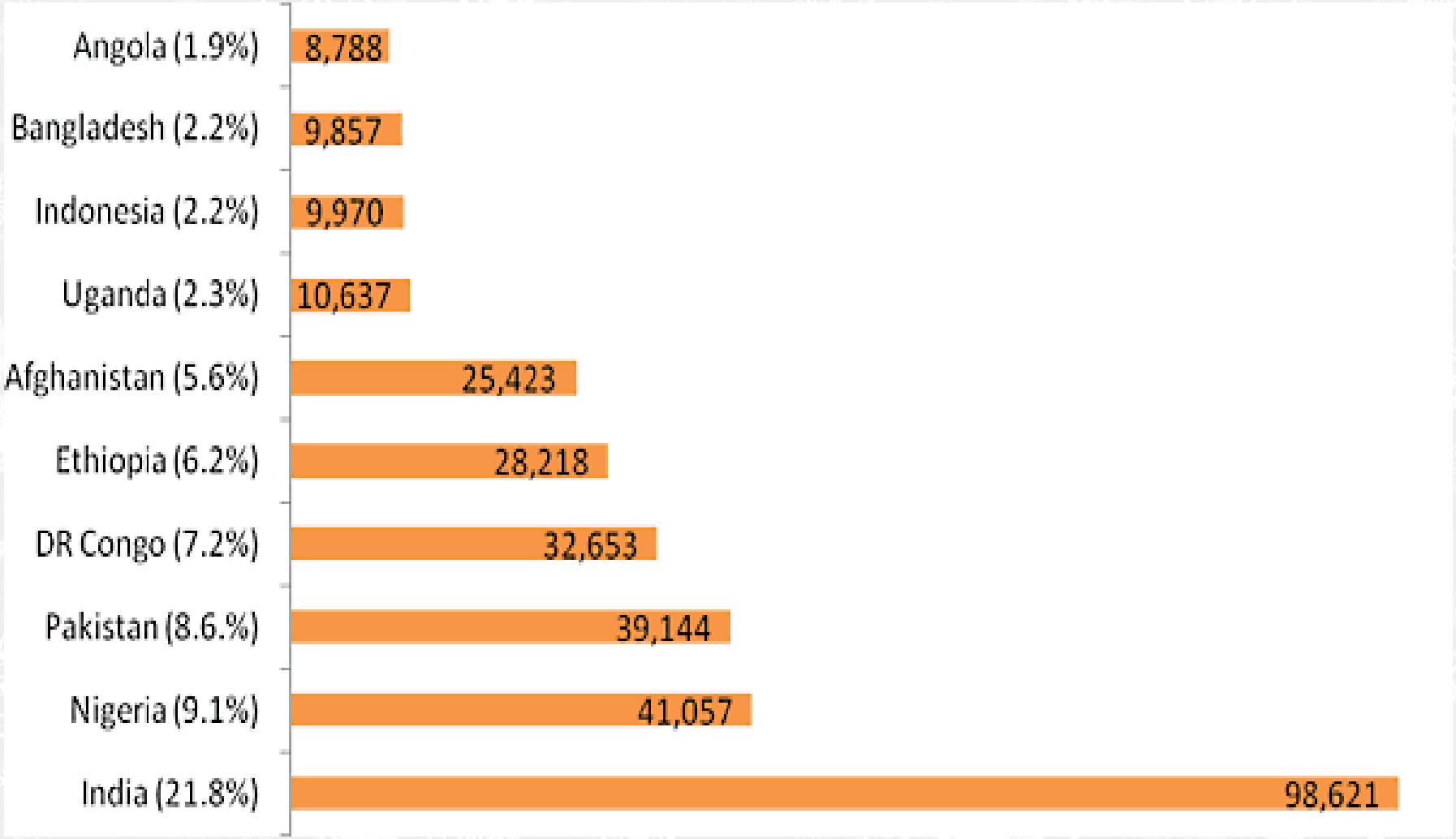
Data Source: WHO/IVB Rotavirus diseases burden estimates, January 2012

Map production: Immunization Vaccines and Biologicals, (IVB), World Health Organization

Date of slide: 02 February 2012

WHO

Rotavirus


Angola (1.9%)
8,788
Bangladesh (2.2%)
9,857
Indonesia (2.2%)
9,970
Uganda (2.3%)
10,637
Afghanistan (5.6%)
25,423
Ethiopia (6.2%)
28,218
DR Congo (7.2%)
32,653
Pakistan (8.6.%)
39,144
Nigeria (9.1%)
41,057
India (21.8%)
98,621

Rotavirus

- En sık görüldüğü aylar aralık ve ocak
- Fekal-oral yolla yayılır
- Rotavirüs çoğunlukla dehidratasyonla seyreden sulu ishal, bulantı, kusma ve karın ağrısına neden olur
- Her yaşta görülebilirse de en sık 6-24 aylık çocukları etkiler
- Üç aydan küçük çocuklar transplasental antikor ve muhtemelen emzirme ile korunurlar

Parashar UD. Emerg Infect Dis 2003;9: 565–72.

Rotavirus bulaş

- kontamine su ve gıda
- dışkı ile kirlenmiş eller
- çeşitli eşyalar ve oyuncaklar
- kişiden kişiye direkt ya da indirekt temas

Parashar UD. Emerg Infect Dis 2003;9: 565–72.

Research letters

Emerging group-A rotavirus and a nosocomial outbreak of diarrhoea

Marc-Alain Widdowson, Gerard J J van Doornum, Wim H M van der Poel, Annette S de Boer, Ulrike Mahdi, Marion Koopmans

A P[6]G9 group-A rotavirus caused a protracted hospital outbreak of neonatal diarrhoea in the Netherlands. The outbreak lasted 5 months with 52 cases and an average attack rate of 40%, 46 cases were in an incubator section for neonates under 1 month of age. Rotavirus P161G9 was detected by RT-PCR in stool samples from the 31 cases tested. Emergence of this genotype in Europe may have implications for neonates lacking protective maternal antibodies and for the development of rotavirus vaccines.

Rotavirus infections are a major cause of severe diarrhoea in infants and young children all over the world. Illness is most severe in children between 6 months and 2 years of age, whereas neonatal rotavirus infections are generally symptomless, probably due to maternal antibody protection.[1] Two viral surface proteins, P and G, are thought to be immunologically important since they induce type-specific neutralising antibodies. Many different variants have been described, but in Europe and the USA most strains belong to the genotypes P[4]G2, or combinations of P[8] with G1, 3, or 4.[1] Therefore, many candidate rotavirus vaccines are based on the G1–4 strains. We describe a reported outbreak of a genotype P[6]G9 rotavirus that caused neonatal diarrhoea in a paediatric ward in the Netherlands.

To test for subclinical infection, ten symptomless neonates that were nursed in the incubator section alongside neonates with rotavirus illness were tested for rotavirus infection by RT-PCR. One neonate tested rotavirus positive and nine were negative.

Rotavirus genotype P[6]G9 has been increasingly reported in the past few years from surveillance data in more-developed and less-developed countries. In Bangladesh, P[6]G9 (first detected in 1995) was among the most common strains in 1996 and 1997.[2,3] Similarly, in a report earlier this year, genotype P[6]G9 strains were described in the UK.[4] This genotype, however, has never previously been reported to cause outbreaks.

We show that rotavirus P[6]G9 can cause serious nosocomial outbreaks of diarrhoea in neonates. Infections of rotavirus in neonates are often symptomless,[1] but in this outbreak a high proportion of neonates exposed to the incubator section had clinical signs and only one symptomless infection was detected. This high attack rate may, in part, be due to the lack of protective antibodies in a high proportion of neonates, as P[6]G9 rotaviruses have not previously been found in the Netherlands[5] and therefore mothers may not have been exposed to this genotype.

Rotavirus

- Yaklaşık **2-4 günlük inkübasyon dönemi** sonrasında aniden ateş ve kusma, ardından sayısı 10'a ulaşan ishal gelişir
- Gaita kansız ve bol suludur, kan ve lökosit nadiren, mukus %20 oranında görülür
- Hastalık **4-8 günde** kendi kendini sınırlar. Genellikle tam iyileşme olur
- Rotavirüse bağlı ölümler **dehidratasyon** ve **elektrolit** bozukluğuna bağlıdır

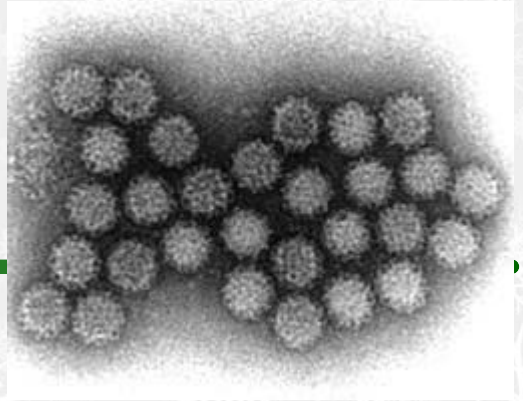
Bernstein DI, Ward RL. Rotaviruses. In: Feigin RD, Cherry JD, Demmler GJ, Kaplan SL, (eds). Textbook of Pediatric Infectious Diseases. Philadelphia 5th edition. Saunders. 2004: 2110-2133

Vesikari skorlama sistemi

İshal süresi (gün) Puan	Vücut ısısı (axiller) Puan
1-4 1	36.6 -.37.9 1
5 2	38 - 38.4 2
≥6 3	≥38.5 3
İshal sayısı / gün	**Tedavi**
1-3 1	Rehidratasyon 1
4-5 2	Hastaneye yatırma 2
≥ 6 3	
Kusma süresi (gün)	**Dehidratasyon**
1 1	% 1-5 2
2 2	≥ % 6 3
≥ 3 3	
Kusma maksimum sayısı/ gün	
1 1	
2-4 2	
≥ 5 3	

Ruuska T and Vesikari T. Scand J. Infect Dis. 1990; 22: 259-267.

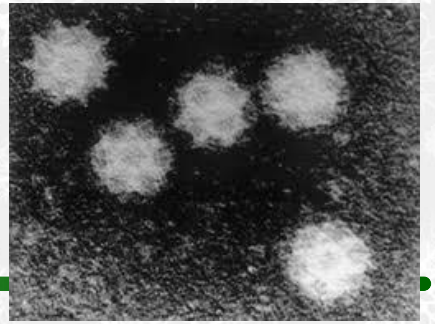
İnsan calisivirus



- Calicivirüsler 27-35 nm çapında, tek sarmallı RNA virüsleridir
- İnsan calisivirusları iki cinse ayrılır: **Sapovirus** ve **Norovirus**
- Norovirus'un prototipi Norwalkvirus
- Eskiden Norwalk benzeri virus olarak adlandırılıyordu)
- Sapovirus'da klasik calisivirus olarak kabul edilir

Zheng DP. J Clin Microbiol 2009;48:168–177.
Zheng DP. Virology 2006;346:312–323.

İnsan calisivirus



- İnsan calsivirusları tüm dünyada yaygındır
- Sero-sürveyans çalışmaları çocukların NLV'lere karşı antikorları erken yaşlarda edindiklerini ve antikor prevalansının okul yılları boyunca erişkin döneme ulaşıncaya kadar giderek arttığını göstermektedir
- Seroprevalans gelişmekte olan ülkelerde daha yüksektir

Jiang X. J Gen Virol 1995; 76: 2739–2747

İnsan calisivirus

- Kalabalık topluluk halinde bulunan yerlerde, bakımevlerinde, aile içinde çabuk yayılır
- Yüzme havuzu veya yiyeceklerden kaynaklanan salgınlar yapabilirler
- Nazokomiyal yayılım olabilir
- Hastalık bütün mevsimlerde görülebilir
- Bulaşma fekal-oral yolla olur

Norovirus

- Endemik ishallerde ve salgınlarda sık görüldüğü düşünülen bir virus
- ABD'de yılda 21 milyon vaka
- Global olarak her yıl yıl 5 yaş> 218.000 ölüm ve 1.1 milyon yatış
- Beş yaş altında ağır ishallerin %12'sinden sorumlu olduğu tahmin ediliyor
- Çocuklarda da toplum kökenli ishalin en sık sebebi olabilir. Ancak daha az ağır seyredip hospitalizasyona sebep olmadığından tanı konulamıyor

Phillips G. Am J Epidemiol 2010;171:1014–1022.
Patel MM. Emerg Infect Dis 2008;14:1224–1231

Norovirus

- Norovirus 5 farklı genogruba ayrılır (I-V) (I,II,IV insanda hastalık yapar)
- Genogrup I ve II'nin 8 ve 10 genotipi identifiye edilmiştir
- Son yıllarda ishal salgınlardan genogrup II genotip 4 sorumlu tutulmuşur

Phillips G. Am J Epidemiol 2010;171:1014–1022.
Patel MM. Emerg Infect Dis 2008;14:1224–1231

Norovirus

- Eskiden besin kaynaklı bulaş primer iken, artık kişiden kişeye bulaş önemli
- Hastaneler, restoranlar, bakımevleri, gemi turları, uçaklar salgınların sıklıkla görüldüğü yerler
- Restoranda salgınlarının en sık sebebi, enfekte aşcıların elleriyle pişmemiş yiyeceklere dokunmasıdır
- ABD'de besin kaynaklı bulaşın en sık sebebi
- ABD'de ekonomik yükü yılda 500 milyon dolar

Phillips G. Am J Epidemiol 2010;171:1014–1022.
Patel MM. Emerg Infect Dis 2008;14:1224–1231

Sapovirus

- Küçük çocuklarda sporadik vakalarla ilişkili
- İngiltere ve Finlandiya'da gastroenterit epizotlarının %10'ından sorumlu ve Finlandiya'da 2 yaşın altında hospitalizasyonların %4'inden sorumlu
- Norovirus enfeksiyonundan daha hafif seyreder ve salgınlar daha çok erişkinlerde görülür

Pang XL. Pediatr Infect Dis J 1999;18:420–426.

Comparison of Clinical Features of Childhood Norovirus and Rotavirus Gastroenteritis in Taiwan

Tzee-Chung Wu, Hsioa-Hui Liu, Yann-Jang Chen, Ren-Bin Tang, Be-Tau Hwang, Han-Chih Yuan*

Children's Medical Center, Taipei Veterans General Hospital and National Yang-Ming University, Taipei, Taiwan, R.O.C.

Background: Viral gastroenteritis is a common acute infectious disease in infants and young children. This study compared the incidence and clinical features of childhood norovirus (NV) and rotavirus (RV) gastroenteritis in Taiwan.

Methods: Stool specimens were collected from children with acute gastroenteritis aged 6 months to 14 years who were treated at the Children's Medical Center of Taipei Veterans General Hospital between January 2004 and March 2005. The incidence, clinical manifestations, and laboratory findings of childhood NV gastroenteritis were analyzed and compared with those of patients with RV gastroenteritis. Patients with underlying diseases associated with diarrhea or those diagnosed with bacterial gastroenteritis were excluded. Stool specimens were tested for NV and RV using enzyme immunoassay (EIA). NV genogroups were determined by reverse-transcriptase polymerase chain reaction.

Results: Among the 201 patients included in this study, NV was detected in 44 (21.9%) by 1 or more tests (22 by EIA). Five of these isolates were genogroup I (11.3%), and 39 were genogroup II (88.7%). Fifty-two (25.9%) specimens had a positive EIA result for RV. Compared with NV, patients with RV gastroenteritis had a significantly higher percentage of diarrhea (94 *vs.* 69%, $p < 0.001$), fever (82 *vs.* 26.2%, $p < 0.001$), and longer hospital stay (3.81 *vs.* 2.93 days, $p = 0.048$). Laboratory studies showed significantly higher liver enzymes and C-reactive protein levels in patients with RV infection. In contrast, white blood cell counts were significantly higher in patients with NV infection.

Conclusion: Norovirus is one of the leading agents of acute gastroenteritis in children in Taiwan, and genogroup II is the predominant type. [*J Chin Med Assoc* 2008;71(11):566–570]

Key Words: gastroenteritis, genogroup, norovirus, rotavirus

Recurring Norovirus Transmission on an Airplane

Craig N. Thornley,[1] Nicola A. Emslie,[2] Tim W. Sprott,[2] Gail E. Greening,[3] and Jackie P. Rapana[1]

[1]Auckland Regional Public Health Service, Auckland District Health Board, Auckland, New Zealand; [2]Air New Zealand Medical Unit, Air New Zealand, Auckland, New Zealand; and [3]Environmental Health Food Group, Institute of Environmental Science and Research, Porirua, New Zealand

(See the Editorial Commentary by Lopman, on pages 521-22.)

Background. Previously reported outbreaks of norovirus gastroenteritis associated with aircraft have been limited to transmission during a single flight sector. During October 2009, an outbreak of diarrhea and vomiting occurred among different groups of flight attendants who had worked on separate flight sectors on the same airplane. We investigated the cause of the outbreak and whether the illnesses were attributable to work on the airplane.

Methods. Information was obtained from flight attendants on demographic characteristics, symptoms, and possible transmission risk factors. Case patients were defined as flight attendants with diarrhea or vomiting <51 hours after the end of their first flight sector on the airplane during 13–18 October 2009. Stool samples were tested for norovirus RNA.

Results. A passenger had vomited on the Boeing 777-200 airplane on the 13 October flight sector. Sixty-three (82%) of 77 flight attendants who worked on the airplane during 13–18 October provided information, and 27 (43%) met the case definition. The attack rate among flight attendants decreased significantly over successive flight sectors from 13 October onward ($P < .001$). Working as a supervisor was independently associated with development of illness (adjusted odds ratio, 5.8; 95% confidence interval, 1.3–25.6). Norovirus genotype GI.6 was detected in stool samples from 2 case patients who worked on different flight sectors.

Conclusions. Sustained transmission of norovirus is likely to have occurred because of exposures on this airplane during successive flight sectors. Airlines should make provision for adequate disinfection of airplanes with use of products effective against norovirus and other common infectious agents after vomiting has occurred.

Norovirus is considered to be the most frequent cause of gastroenteritis in developed countries [1, 2] with a recently estimated community incidence of 4.5 cases per 100 person-years [3]. Although transmission of norovirus is ultimately through oral ingestion of virus shed by infected individuals in feces [4] or vomit [5], several features of the virus facilitate its spread [6]: the virus is shed before and after illness, persists in the environment, exhibits considerable strain diversity, is genetically labile, and requires only a low infectious dose.

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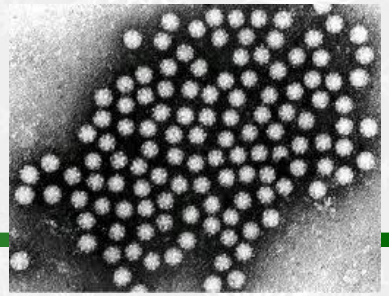
Clinical Infectious Diseases 2011;53(6):515–520

1058-4838/2011/536-0003$14.00
DOI: 10.1093/cid/cir465

Outbreaks of norovirus gastroenteritis not related to contaminated food or water have occurred in many different settings, including cruise ships [7], hotels [8], public venues [9], nursing homes [10], and hospitals [11]. Features of these settings that may predispose to norovirus transmission are large numbers of individuals, close personal contact, and shared sanitation facilities. These features are also present in airplane cabins; however, despite the global volume of air travel, relatively few norovirus outbreaks on airplanes have been reported [12–16]. Of those that have, all involved a single flight sector, defined as the period from an airplane's departure from one airport to arrival at the next airport.

On 18 October 2009, an airline medical team became aware that multiple flight attendants working in different teams had become unwell with gastroenteritis since 14 October. All these teams had worked on a single airplane over successive flight sectors. The airline medical

Astrovirus



- İlk olarak 1975 yılında tanımlanan astrovirüsler zarfsız, 28-30nm çapında, tek sarmallı RNA virüsleridir
- Elektron mikroskopide karakteristik olarak beş veya altı uçlu yıldız şeklinde görünürler
- Tek sarmallı RNA genomu üç yapısal protein kodlar ve yaklaşık 7,2 Kb uzunluğundadır
- Virüsde 8 farklı serotip (1-8) tanımlanmıştır. İnsanlarda genellikle serotip 1 hastalık yapar

Madeley CR. Lancet 1975;2:451–452.
Noel JS. J Clin Microbiol 1995;33:797–801.

Astrovirus

- Astrovirüs dünyanın hemen her yerinde ishale yol açan, okullar, hastanelerin çocuk servisleri, yaşlı bakım evleri ile çocuk bakım merkezlerindeki ishal salgınlarından sorumlu tutulan, bu nedenle de kişiden kişiye bulaştığı düşünülen bir virüstür
- Hastalık özellikle dört yaşından küçük çocuklarda ve yaşlılarda görülür.
- Erişkinlerin %80'inden çoğunda bu virüse karşı antikor vardır
- Yıl boyunca görülebilirse de, astrovirüs ishallerinin en sık ortaya çıktığı dönem kış-ilkbahar ayları
- Fekal-oral yolla bulaşır .
- Astrovirüs gıdalarla ilgili salgınlarla da ilişkili bulunmuştur

Mitchell DK. Pediatr Infect Dis J 1999;18:249–254.

Astrovirus klinik

- Asemptomatik enfeksiyon sıktır
- Astrovirüslerin neden olduğu ishal genellikle çocuklar ve yaşlılarda
- İnkübasyon dönemi 1-4 gündür
- Semptomlar arasında genellikle dört gün süren düşük derecede ateş, kırıklık, bulantı, kusma ve sulu ishal bulunur
- Kusma diğer virüslerle olduğundan daha seyrektir ve klinik rotavirusdan daha hafif seyirlidir

Adenovirus

- Zarfsız ve çift sarmal DNA'ları olan 70-80 nanometre (nm) çapında, ikozohedral virüslerdir
- Adenovirusların 51 farklı serotip içeren 6 alttür (A-F) insanlarda hastalık yaparken, alttür F (40 ve 41) gastroenterit yapar
- Özellikle tipik üst solunum yolu enfeksiyonları esnasında ve sonrasında dışkıda birçok adenovirüs serotipi bulunursa da sadece serotip 40, 41 ve nadiren de 31 gastroenterit yapar

de Jong JC. J Med Virol 1983;11:215–231.
Brown M,. Arch Virol Suppl 1996;12:301–307.

Adenoviruslar

Farklı tipler belirli bir hastalıkla ilişkilidir.

Grup	Doku tropizmi	Serotipler
A	Gastrointestinal yol	12, 18, 31
B	Üriner yol, Resp.yol	3, 7, 11, 14, 16, 21, 34, 35, 50
C	Üst respiratuvar yol	1, 2, 5, 6
D	Göz, gastrointestinal yol	8-10, 13, 15, 17, 19, 20 22-30, 32, 33, 36-39, 42-49, 51
E	Respiratuvar yol, göz	4
F	Gastrointestinal yol	40,41

Adenovirus

- Enterik adenovirüs tiplerinden 40 ve 41 çok yaygın olup endemik ishale ve hastanelerde, yetimhanelerde ve çocuk bakım merkezlerinde ishal salgınlarına yol açar
- Antikor prevalans çalışmaları çocukların %50'sinden fazlasının üç veya dört yaşına gelene kadar seropozitif hale geldiklerini göstermektedir
- Enfeksiyon tüm yıl boyunca ortaya çıkabilir. Mevsimsel ilişkisi yoktur.
- Bulaşma oral-fekal yolla olup, inkübasyon süresi 3 ila 10 gündür.
- Hastalık 5-12 gün sürer. Serotip 40'a bağlı ishalin süresi ortalama 8,6 gün, serotip 41'e bağlı ishal süresi ortalama 12,2 gündür

Kim KH. J Clin Microbiol 1990;28:2279–2284.
Yolken RH. J Pediatr 1982;101:21–26.

Adenovirus

- Enterik adenovirüsler kusma ve ateşle birlikte olabilen ve 6-9 gün kadar süren ishale neden olur. İshal, kan veya dışkıda lökosit içermeyen sulu bir ishaldir. Uzun süren laktoz intoleransı gelişebilmektedir.
- Çoğu hastada dehitratasyon şiddetli olmaz. İshalin süresi rotavirüs ishallerine göre daha uzundur.
- Sıklıkla 2 yaşın altındaki çocuklarda görülür, erişkinlerde sık görülmez
- Enterik adenovirüsler hastanede yatan çocuk ishal vakalarının %5 ila %10'sine neden olurlar . Ve hastane kökenli ishallerin sık sebebi
- Toplumda ishallerin%1-4 sebebi
- Asemptomatik enfeksiyon sıktır, hastalığın ardından virüs atılımı haftalarca sürebilir

Kotloff KL. Pediatrics 1989;84:219–225.

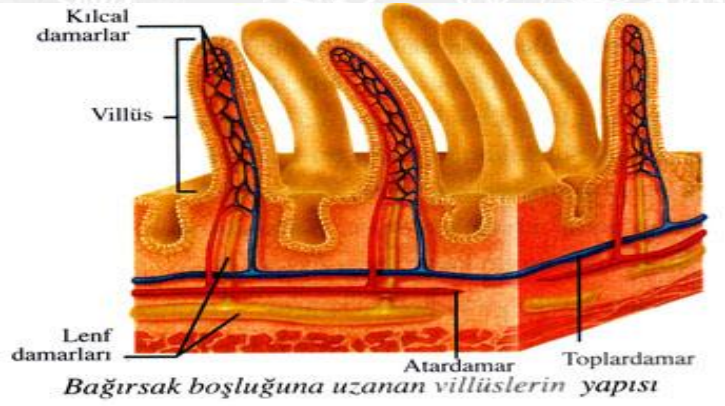
VİRAL GASTROENTERİTLERİN EPİDEMİYOLOJİK ÖZELLİKLERİ					
Özellik	**ROTAVIRÜS**	**NOROVIRÜSLER**	**SAPOVIRÜSLER**	**ASTROVIRÜSLER**	**ADENOVIRÜSLER**
Görülme yaşı	< 5 yaş	Tüm yaşlar	< 5 yaş	< 2 yaş	< 2 yaş
Bulaş yolu	Insandan insdan fekal- oral yolla ya da cansız objelerle	insandan insana fekal- oral yolla, cansız objelerle, su/besinlerle	insandan insana fekal- oral yolla	insandan insana fekal- oral yolla	insandan insana fekal- oral yolla
Inkübasyon süresi	1-3 gün	12-48 saat	12-48 saat	1-4 gün	3-10 gün
SEMPTOMLAR					
İshal	Bol, sulu (5-10 kez/gün)	Ani başlangıçlı, sulu	Sulu; Rotavirüs'ten daha hafif	Sulu; Rotavirüs'ten daha hafif	Sulu; Rotavirüs'ten daha hafif; biraz daha uzayabilir
Kusma	% 80-90	>%50 baskın semptom	Rotavirüs'e göre daha nadir	Rotavirüs'e göre daha nadir	Rotavirüs'e göre daha nadir
Ateş	Sık	Nadir, genelde daha hafif	Nadir, genelde daha hafif	Nadir, genelde daha hafif	Nadir, genelde daha hafif
Hastalık süresi	2-8 gün	1-5 gün	1-4 gün	1-5 gün	3-10 gün
BAŞLICA TANI YÖNTEMİ	Gaita EIA ya da LPA	RT-PCR	RT-PCR	Gaita EIA	Gaita EIA

EIA: Enzim immunoassay

LPA: Lateks parça agglütinasyonu

RT-PCR: Reverse transcriptase polimeraz zincir reaksiyonu

Viral Gastroenterit Patogenez

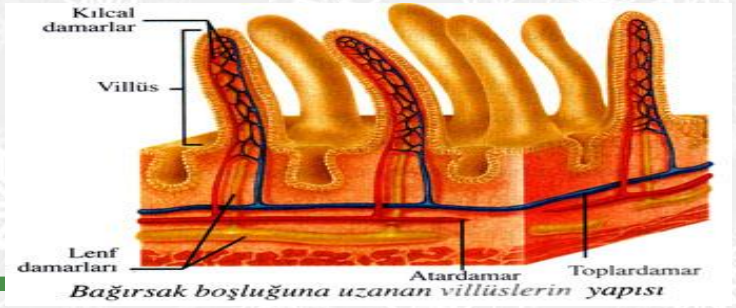


Bağırsak boşluğuna uzanan villüslerin yapısı

- Sıklıkla yakın temas sonucu **fekal-oral** yolla bulaş olur
- Ancak norovirus kolaylıkla **kontamine yiyecek ve suyla** bulaşır. Bu yüzden **besin kaynaklı** bulaşda major bir etken
- Hasta kişilerin kusmuğunda norovirus bulunur. Bu yüzden **damlacık yoluyla** hastanede ve uçaklarda bulaşda önemli
- Adenovirusda bulaş tam anlaşılmamış. Ancak fekal-oral yolla olduğu tahmin ediliyor
- **Fomitlerle** bulaş önemli olabilir

Kapikian AZ. 1993;269:627–630.
Widdowson MA.Emerg Infect Dis 2005;11:95–102.
Marks PJ. Epidemiol Infect 2000;124:481–487.

Viral Gastroenterit Patogenez



- İnce barsağın villüs epitelinin uç noktalarında kopyalanarak zedelenmeye yol açar ve villüslerin kısalmasına neden olur
- Rotavirus abzortif enterosit kaybına yol açarak osmotik ishal ve kalsiyum kanallarını açarak sekretuar ishale neden olabilir
- Normal şartlarda absorbsiyonun gerçekleştiği villüs epitelinin kaybı sonucu uygun olmayan şekilde su ve elektrolit sekresyonuna neden olur
- Ayrıca villüs hasarı disakkaridaz enzimlerinin de eksilmesine neden olarak geçici disakkaridaz, özellikle de laktaz eksikliğine neden olabilir

Mavromichalis J. Arch Dis Child 1977;52:589–591.
Ball JM. Science 1996;272:101–104.

Viral Gastroenterit Patogenez

- Akut enfeksiyon sırasında dışkıda bol miktarda virus bulunur
- Ancak rotavirus, norovirus ve sapovirus hastalıktan 1-2 gün önce ve hastalık bittikten sonra 7 gün boyunca daha atılabilir. Buda bulaşda önemli
- Özellikle norovirusda asemptomatik enfeksiyon sık. Bulaşda yeri?

Kapikian AZ. JAMA 1993;269:627–630.

Viral Gastroenterit Patogenez

- Rotavirusa karşı edinilmiş immünite söz konusu. Tam bir korunma multiple enfeksiyonlardan sonra olur
- Primer enfeksiyondan sonra homotipik immünite güçlüdür, ancak tekrarlayan enfeksiyonladan sonra diğer suşlara karşıda immünite genişler
- Norovirüs enfeksiyonlarına karşı gelişen immünite kısa süreli olup (aylar-1 yıl), heterotipik immünite gelişimi sınırlı olduğundan; büyük çocuklar ve erişkinlerde de enfeksiyon görülür.

Velazquez FR. J Infect Dis 2000;182:1602–1609.
Parrino TA. N Engl J Med 1977;297:86–89.

Viral gastroenterit klinik

- Kısa bir inkübasyon periyodundan sonra akut ishal başlar
- Klinik farklı viruslerde ayırt edilemez
- Kusma rotavirusda erken bir bulgu iken, norovirusda da görülür
- İshal genellikle suludur kan ve görülebilir mukus olmaz
- Çocukların yarısında erken bir bulgu olan ateş olur
- Ateş ve kusma 1-3 gün içerisinde sonlanır, ishal özelliklede rotavirusda ısrarla devam eder
- Diğer bulgular karın ağrısı ve kırgınlık

Kaplan JE. Am J Public Health 1982;72:1329–1332.

Viral gastroenterit klinik

- En önemli komplikasyon elektrik anormalliğinin eşlik ettiği dehidratasyon
- Haftalarca süren malabsorbsiyon gelişebilir
- Solunum sistemi bulguları varsa, kışın eşlik eden başka bir etkene bağlı olabilir
- Ekstraintestinal komplikasyonlar nadirdir. Ancak ensefalit, hemofagositik lenfohistiyositoz, akut miyozit, akut flask paralizi ve ani bebek ölüm sendromu nadiren rotavirus enfeksiyonu olan çocuklarda tanımlansada, bağlantısı kesin değil
- İmmünyetmezlikli kişilerde klinik bulgular daha uzun sürer

Lopman BA. Clin Infect Dis 2004;39:318–324.

Scandinavian Journal of Infectious Diseases, 2009; 41: 857–861

informa
healthcare

ORIGINAL ARTICLE

Bacteraemia and candidaemia: A considerable and underestimated complication of severe rotavirus gastroenteritis

ERGİN ÇİFTÇİ, ANIL TAPISIZ, HALİL ÖZDEMİR, HALUK GÜRİZ, TANIL KENDİRLİ, ERDAL İNCE & ÜLKER DOĞRU

From the Department of Paediatric Infectious Disease, Ankara University Medical School, Ankara, Turkey

Abstract

Despite the high incidence of rotavirus gastroenteritis, secondary bacteraemia later in the course of the disease has rarely been reported. To date, the exact incidence of this complication has not been determined. A prospective study was conducted between January 2007 and December 2008 to determine the incidence of bacteraemia by organisms of the normal intestinal flora during severe rotavirus gastroenteritis. Rotavirus gastroenteritis was diagnosed by antigen detection in stool. A previously described 20-point numerical score system was used to determine the severity of disease. There were 289 cases (30%) of rotavirus gastroenteritis during the study period, 106 (36.7%) of which were accepted to be severe rotavirus gastroenteritis and hospitalized. On admission stool and blood cultures tested negative. In cases of persistent or recurrent fever, additional blood cultures were obtained. Among cases with severe rotavirus gastroenteritis, 4 (3.8%) had positive blood cultures (Klebsiella pneumoniae in 1 patient, Escherichia coli in 1 patient, Pseudomonas aeruginosa and Candida albicans in 1 patient, and Candida albicans in 1 patient). All patients were successfully treated with fluid replacement and antimicrobial therapy. Bacteraemia and candidaemia appear to be a considerable and underestimated complication of severe rotavirus gastroenteritis.

Benign Afebrile Convulsions in the Course of Mild Acute Gastroenteritis

A Study of 28 Patients and a Literature Review

Wael Fasheh Youssef, MD, Rosa Pino Ramírez, MD,* Jaume Campistol Plana, MD, PhD,† and Mercedes Pineda Marfa, MD, PhD†*

Objectives: Since the description of afebrile convulsions in the course of mild acute gastroenteritis (AGE) in 1982 by Morooka in Japan, there have been few reports of further cases outside Asia. The aim of this study was to share our casuistry—from a non-Asian country.

Methods: This is a retrospective study of identified cases in our center from January 2002 to December 2007.

Results: A total of 28 patients were studied. All were previously healthy patients who experienced convulsions with mild AGE without dehydration and with normal blood analysis. The mean age was 17.25 months (range, 6–48 months), with 93% younger than 24 months. Seizures were generalized tonic-clonic (61%), followed by generalized tonic (31%), and hypotonic (5.2%), with 2 (2.6%) partial. Only 8 patients (28.6%) presented one convulsion, and in 13 patients (46%), the seizures were in clusters from 3 to 6. Eleven patients (39%) presented 2 different types of convulsion. The duration of the crises ranged from 30 seconds to 10 minutes, and all of them occurred within 24 hours of the first. Electroencephalograms, obtained for all patients, were normal. Rotavirus was the main infectious agent in the AGEs, found in 11 patients with 22 determinations. In one patient, *Salmonella* serotype Enteritidis was isolated. All of the patients developed favorably, with no sequelae or epilepsy during the follow-up period.

Conclusions: Afebrile convulsion in the course of mild gastroenteritis exists in our environment. It is a banal symptom in the course of the disease with good prognosis. Recognition of this fact may help avoid needless explorations and treatment in patients of this kind.

Key Words: afebrile convulsions, mild gastroenteritis, rotavirus

(*Pediatr Emer Care* 2011;27: 1062–1064)

clustered. Studies carried out yield normal results in the following: acid-base balance, glucose, ionogram, cerebral spinal fluid, electroencephalography, and neuroimaging. Subsequent recovery from the episode is complete without neuropsychic deterioration. The incidence of this clinical symptom outside Asia is relatively unknown. It would be interesting to learn more about its characteristics, in that the convulsions are benign and require no additional investigation or treatment.

With the aim of learning more about this kind of incident and the clinical characteristics of banal afebrile convulsions in the course of mild AGE in our environment, we carried out a retrospective study of those cases that may be included in this pathologic finding.

METHODS

This was a retrospective study of the cases coded and on file at our center (Hospital Sant Joan de Déu in Barcelona, a third-level hospital that attends to 65,000 pediatric emergencies per year), some with gastroenteritis and convulsions in the same admission, during the years 2000 to 2007. Data were collected on those patients who fulfilled the inclusion criteria: (1) convulsions accompanied by gastrointestinal symptoms; (2) absence of dehydration or mild dehydration (<5%); (3) temperature lower than 37.5°C before and after the episode until resolution of the intestinal symptoms; (4) normal study results for hemogram, acid-base balance (pH 7.25–7.4, standard bicarbonate >18, excess of base greater than −10), sodium, potassium, calcium, and glucose level greater than 60 mg/dL; and (5) no history of administration of antiemetics in the intestinal process. Excluded

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Benign Convulsions With Mild Gastroenteritis: Is It Associated With Sodium Channel Gene *SCN1A* Mutation?

Wen-Chin Weng, MD[1], Shinichi Hirose, MD, PhD[2], and Wang-Tso Lee, MD, PhD[1]

Abstract

Benign convulsions with mild gastroenteritis were afebrile seizures associated with gastroenteritis in previously healthy infants or young children. It has been thought to be a continual spectrum of benign infantile convulsions because of overlapping clinical pictures. Recently, molecular genetic studies have suggested a channelopathy in benign infantile convulsions. The authors prospectively studied the clinical features of benign convulsions with mild gastroenteritis in Taiwanese children and clarified the relationship between neuronal sodium channel alpha 1 subunit (*SCN1A*) gene and benign convulsions with mild gastroenteritis. The clinical pictures in their patients were similar to those of previous studies except for the low rate of positive rotavirus antigen in the stool, which may indicate a season-related viral infection. No mutations in the *SCN1A* gene were identified in all patients. This study suggested that *SCN1A* mutations are probably not associated with benign convulsions with mild gastroenteritis. Other possible pathogenic mechanisms need to be researched in the future.

Keywords

convulsions, gastroenteritis, *SCN1A*, Taiwan

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Benign convulsions with mild gastroenteritis have been reported in Japan and other countries.[1-4] It is well recognized in Japanese pediatric patients and is characterized as a cluster of afebrile seizures associated with gastroenteritis in previously healthy infants or young children.[1-5] The laboratory examinations including electrolytes, blood glucose, and cerebrospinal fluid are normal and interictal electroencephalogram shows no abnormalities. The seizures often occur in clusters and are resistant to anticonvulsants such as diazepam or phenobarbital.[2,6] However, the prognosis is considered excellent despite initially intractable seizures, and psychomotor development is normal before and after the episodes.[2,4]

The pathogenic mechanisms of benign convulsions with mild gastroenteritis remain unclear. Because the clinical features of benign convulsions with mild gastroenteritis resemble those of benign infantile convulsions, benign convulsions with mild gastroenteritis have been classified into the spectrum of benign infantile convulsions with an underlying genetic predisposition.[7-11] Recent molecular genetic studies have suggested a channelopathy in benign infantile convulsions.[12-15] Furthermore, de novo mutations of the sodium channel gene *SCN1A* were identified in alleged vaccine encephalopathy.[16] Whether the *SCN1A* mutation is also a responsible mechanism for benign convulsions with mild gastroenteritis remains to be clarified. In the present study, we prospectively investigated the clinical features of benign convulsions with mild gastroenteritis in children in Taiwan, and performed the mutation analysis of the *SCN1A* gene in these patients to clarify the relationship of benign convulsions with mild gastroenteritis with *SCN1A* mutations.

Subjects and Methods

From January 2006 to December 2006, a total of 12 patients who were admitted to the National Taiwan University Hospital and had

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Norovirus as cause of benign convulsion associated with gastro-enteritis

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Aim: Rotavirus and norovirus gastro-enteritis (GE) are common in children. Complications, except severe dehydration, are rare. Rotavirus was known to cause seizures and even GE encephalopathy, but these complications are less described in norovirus infection. The objective of this study is to compare the demographic features, clinical manifestations including the incidence of afebrile seizure, and the outcomes in children with rotavirus and norovirus infections.

Methods: This is a retrospective review of children between age 1 month and 6 years admitted to the paediatric department of a regional hospital in Hong Kong with rotavirus and norovirus infections over a period of 3 years from 1 June 2006 to 31 May 2009. Their demographic data, clinical features, laboratory results and outcomes were compared and analysed.

Results: Two hundred and thirty-two children with rotavirus and 173 children with norovirus GE were admitted within the study period. Afebrile seizure commonly occurred in norovirus infection (8.67% vs. 1.29%, $P < 0.001$). Children with rotavirus infection had higher temperature and more diarrhoea episodes, while more blood-stained stool was noted in the norovirus group. Rotavirus-infected patients stayed longer in hospital. All of them had full recovery without any complication. Among the 18 patients who developed afebrile convulsions, 17 of them had neuroimaging performed, which was normal. Fourteen of them had electroencephalogram (EEG) performed, demonstrating normal or non-specific findings. None of them developed subsequent seizure attack after the GE episode.

Conclusions: Norovirus is more commonly associated with benign convulsion in GE than rotavirus. We need to identify the presence of virus, in particular norovirus, in children with GE and afebrile generalised tonic–clonic seizure. Further neuro-investigations may not be necessary once the aetiology is established. Prognosis is excellent in this group of children and prophylactic anticonvulsant is not needed.

Key words: gastro-enteritis; norovirus; rotavirus; seizures.

What is already known on this topic

1 Benign convulsion associated with mild gastro-enteritis (CwG) can be seen in rotavirus gastro-enteritis.
2 Seizures in CwG are usually multiple in nature.
3 CwG carries a good neurological prognosis.

What this study adds

1 Norovirus is more commonly associated with CwG than rotavirus.
2 Serum biochemistry, neuroimaging and inter-ictal electroencephalogram are usually normal in CwG.
3 Extensive neuro-investigations and long-term anti-epileptic treatment shall be avoided in CwG with normal neurological examination.

Rotavirus and norovirus infections are the most common causes of viral gastro-enteritis (GE) among paediatric patients. During the course of illness, most affected children recover uneventfully, while a small proportion are complicated by seizures. Morooka first reported benign convulsions associated with mild gastro-enteritis (CwG) in Japan in 1982.[1] This was followed by other case report[2] and retrospective studies,[3–6] but they were mostly focused on rotavirus infection. Convulsion associated with norovirus GE is less reported in the literature.

The objective of this study is to compare the patient demographics, clinical manifestations, and the outcomes in children with rotavirus and norovirus infections, with regard to the incidence of afebrile seizures.

Method

This is a retrospective review of children admitted to a regional hospital in Hong Kong for acute GE over a 3-year period from 1st June 2006 to 31st May 2009.

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Conflict of interest statement: Nil to declare.

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Viral gastroenterit salgınında

- Guita örneğinde bakteri veya parazit yoksa
- %50< hastada kusma varsa
- İnkübasyon süresi ortalama 24-48 saat ise
- Hastalık süresi ortalama 12-60 saat ise

Büyük ihtimalle etken norovirus

Lopman BA. Clin Infect Dis 2004;39:318–324.

Viral gastroenterit tanı

- Laboratuvar en iyi tanı; akut hastalık döneminde taze dışkıda **viral antijenin** veya **nükleik asitin** gösterilesi ile konur
- Dışkıda rotavirus antijenin tespit etmeye yarayan ticari kitler kolay ve ucuz metodlardır
- Bunlar VP6 proteinin tespit eden **enzim immunoassay (EIA)** yada **lateks partikül aglütinasyon** testleridir
- Antijen tespit eden yöntemler genel olarak <%90 sensitif ve %95 spesifiktir

Marionneau S. Gastroenterology 2002;122:1967–1977.

Viral gastroenterit tanı

- Rotavirus tespiti ayrıca **elektron mikroskobi**, **virus izolasyonu** ve direkt olarak dışkıdan alınan RNA'da **poliakrilamine jel elektroforezi (PAGE**) ile yapılabilir

- **RT-PCR** yüksek analitik sensitiviteye sahiptir ve virus hastalık yapmadığında da tespit edebilir. RT-PCR klinikte nadiren kullanılır.

- Rotavrus enfeksiyonunun tespiti için **serolojik testler** kullanılabilir ama klinik kullanımda pratik değildir.

Marionneau S. Gastroenterology 2002;122:1967–1977.

Viral gastroenterit tanı

- Calisivirusların tespiti için antijen tespiti yapan kitler mevcut, ancak klinik uygulamada düşük sensitiviteden dolayı kullanımı önerilmemektedir. Ancak salgınlarda faydalı olabilir

- Kalisiviruslar için RT-PCR standart tanı yöntemi olarak yerini alsada klinikte nadiren kullanılır

Duizer E. J Clin Virol 2007;40:38–42.
Gray JJ. Clin Vaccine Immunol 2007;14:1349–1355.

Viral gastroenterit tanı

- Astrovirus antijenini dışkıda tespit edebilen ticari EIA testleri mevcut (yüksek sensitivite ve spesifite)
- Serolojik tetkikler, ve elektron mikroskobi primer olarak araştırmalarda kullanılmaktadır
- Benzer şekilde adenovirusların tespiti için EIA ve lateks aglutinasyon kitleri mevcut olup yüksek spesifiteye ve sensitiviteye sahiptir

King CK. MMWR Recomm Rep 2003;52(RR-16):1–16.

Viral gastroenterit tedavi

- **Spesifik tedavisi yok**

- **Tedavinin temelini**; hastayı hızlıca ve doğru şekilde değerlendirip, sıvı kaybını ve elektrolit bozukluğunu düzeltmek ve hidrasyonunu ve nutriyonunu devam ettirmekdir

Herrmann JE. J Infect Dis 1987;155:1167–1171.

Review®

•Laboratuvara odaklanmamalı, çünkü hastalığın ağır bulguları çıkmadan bozulma olmayacaktır

•Hangi hasta ev gidebilir
•Hangisi gözlenmeli
•Hangisine agresif tedavi

Sy			Severe Dehydration (>9% Loss of Body Weight)
M		restless, irritable	Apathetic, lethargic, unconscious
Thirst	refuse liquids	Thirsty, eager to drink	Drinks poorly; unable to drink
Heart rate	Normal	Normal	tachycardia in
Quality of pulses	Normal	Normal	le
Breathing	Normal	Normal; fa	
Eyes	Normal	Slightly sunken	
Tears	Present	Decreased	Absent
Mouth and tongue	Moist	Dry	Parched
Skin fold	Instant recoil	Recoil in <2 seconds	Recoil in >2 seconds
Capillary refill	Normal	Prolonged	Prolonged; minimal
Extremities	Warm	Cool	Cold; mottled; cyanotic
Urine output	Normal to decreased	Decreased	Minimal

Viral gastroenterit tedavi

- **Tedavi iki kısımdan oluşur:**

Rehidratasyon

İdame tedavisi

- Yeterli glukoz-elektrolit solusyonu içeren oral rehidrasyon (ORS) tedavisi çoğu vakada gerekli olur

Herrmann JE. J Infect Dis 1987;155:1167–1171.

Dehidratasyon derecesine göre tedavi

Degree of Dehydration	Rehydration Therapy	Replacement of Losses	Nutrition
Minimal or no dehydration	Not applicable	<10 kg body weight: 60–120 mL ORS for each diarrheal stool or vomiting episode >10 kg body weight: 120–240 mL ORS for each diarrheal stool or vomiting episode	Continue breastfeeding, or resume age-appropriate normal diet after initial hydration, including adequate caloric intake for maintenance
Mild to moderate dehydration	ORS, 50–100 mL/kg body weight over 3–4 hours	Same	Same
Severe dehydration	Lactated Ringer solution or normal saline in 20 mL/kg body weight intravenously until perfusion and mental status improve; then administer 100 mL/kg body weight ORS over 4 hours or 5% dextrose in ½ normal saline intravenously at twice maintenance fluid rates	Same; if unable to drink, administer through NG tube or administer 5% dextrose in ¼ normal saline with 20 mEq/L potassium chloride intravenously	Same


ALO 171 SİGARA BIRAKMA HATTI MI ?
EVET BUYRUN...
ADRESİ VERİYORUM 2 PAKET BIRAKIRSINIZ
DÜÜTT.....
Şahin GÜNEŞ

ORS tedavisi ve meşrubatların karşılaştırılması

Solution	Carbohydrate (gm/L)	Sodium (mmol/L)	Potassium (mmol/L)	Chloride (mmol/L)	Base* (mmol/L)	Osmolarity (mOsm/L)
ORS						
World Health Organization (WHO) (2002)	13.5	75	20	65	30	245
WHO (1975)	20	90	20	80	30	311
European Society of Paediatric Gastroenterology, Hepatology and Nutrition	16	60	20	60	30	240
Enfalyte®†	30	50	25	45	34	200
Pedialyte®§	25	45	20	35	30	250
Rehydralyte®¶	25	75	20	65	30	305
CeraLyte®**	40	50–90	20	NA††	30	220
Commonly used beverages (not appropriate for diarrhea treatment)						
Apple juice§§	120	0.4	44	45	N/A	730
Coca-Cola®¶¶ Classic	112	1.6	N/A	N/A	13.4	650

*Actual or potential bicarbonate (e.g., lactate, citrate, or acetate).
†Mead-Johnson Laboratories, Princeton, New Jersey. Additional information is available at http://www.meadjohnson.com/products/cons-infant/enfalyte.html.
§Ross Laboratories (Abbott Laboratories), Columbus, Ohio. Data regarding Flavored and Freezer Pop Pedialyte are identical. Additional information is available at http://www.pedialyte.com.
¶Ross Laboratories (Abbott Laboratories), Columbus, Ohio. Additional information is available at http://rpdcon40.ross.com/pn/PediatricProducts.NSF/web_Ross.com_XML_PediatricNutrition/96A5745B11839473852256A80007546E5?OpenDocument.
**Cera Products, L.L.C., Jessup, Maryland. Additional information is available at http://www.ceralyte.com/index.htm.
††Not applicable.
§§Meeting U.S. Department of Agriculture minimum requirements.
¶¶Coca-Cola Corporation, Atlanta, Georgia. Figures do not include electrolytes that might be present in local water used for bottling. Base=phosphate.
Source: Centers for Disease Control and Prevention. Managing acute gastroenteritis among children: oral rehydration, maintenance, and nutritional therapy. *MMWR* 2003;52(No. RR-16):1–16.

Viral gastroenterit tedavi

- Sürekli kusan veya şokun eşlik ettiği ağır dehidrate olan çocuklarda IV rehidrasyon gerekebilir
- Anne sütüne mümkünse devam edilmeli
- Formul mama ile beslenenler rehidrasyonla beraber buna devam etmeliler
- Solid gıda alan çocuklar ishal döneminde buna devam etmeli.
- Yüksek şeker ihtiva eden gıdalardan uzak durulmalı
- Antimikrobiyal tedavi verilmemelidir

Published in final edited form as:
Lancet. 2007 October 6; 370(9594): 1230–1239.

Effect of daily zinc supplementation on child mortality in southern Nepal: a community-based, cluster randomised, placebo-controlled trial

James M Tielsch, **Subarna K Khatry**, **Rebecca J Stoltzfus**, **Joanne Katz**, **Steven C LeClerq**, **Ramesh Adhikari**, **Luke C Mullany**, **Robert Black**, and **Shardaram Shresta**
Department of International Health, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD, USA (Prof J M Tielsch PhD, Prof J Katz ScD, S C LeClerq MPH, L C Mullany PhD, Prof R Black MD); Nepal Nutrition Intervention Project—Sarlahi, Kathmandu, Nepal (S K Khatry MBBS, S C LeClerq, S Shresta MPH); Division of Nutritional Sciences, Cornell University, Ithaca, NY, USA (Prof R J Stoltzfus PhD); and Institute of Medicine, Tribhuvan University, Kathmandu, Nepal (Prof R Adhikari MD)

Summary

Background—Zinc supplementation can reduce subsequent morbidity in children recovering from diarrhoea and respiratory illness in developing countries. However, whether routine supplementation would decrease morbidity and mortality in populations with zinc deficiency is unclear. We assessed the effect of daily zinc supplementation on children in southern Nepal.

Methods—We did a community-based, cluster-randomised, double-masked, placebo-controlled, 2×2 factorial trial in children aged 1–35 months. Treatment groups were placebo, iron and folic acid, zinc, and iron and folic acid with zinc, with daily doses of 12·5 mg iron, 50 μg folic acid, and 10 mg zinc. Study staff gave children tablets on 2 days each week and left tablets with caregivers for other days. All children received vitamin A supplementation twice per year. Results of the iron arm of the trial have been reported previously. Between October, 2001, and January, 2006, 41 276 children were enrolled into the placebo (n=20 308) or zinc (n=20 968) groups and were followed-up for 60 636·3 person-years. The primary outcome was child mortality, and analyses were by intention to treat. Daily reports of signs and symptoms of common morbidities in stratified random subsamples of children were assessed every week for 12 months. This study is registered at ClinicalTrials.gov, number NCT00109551.

Findings—2505 children refused to continue the trial and 3219 children were lost to follow-up. There was no significant difference in mortality between the zinc and placebo groups (316 *vs* 333 deaths; hazard ratio 0·92, 95% CI 0·75–1·12). Zinc had no effect on mortality in children younger than 12 months (181 *vs* 168 deaths; 1·04, 0·83–1·31); mortality was lower, but not statistically significantly so, in older children receiving zinc (135 *vs* 165; 0·80, 0·60–1·06). The frequency and duration of diarrhoea, persistent diarrhoea, dysentery, and acute lower respiratory infections did not differ between the groups.

CONTENTS

Prebiotics and Probiotics in Prevention and Treatment of Infectious Diseases

TREATMENT OF ACUTE

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•Viral ishallerde faydalı olabilir, bakteriyel ishallerde önerilmiyor

The Role of Pre ention and Treatment of C ous Diseases

*Stefan Weichert, M * Horst Schroten, MD,* and Rüdiger Adam, MD*†*

Abstract: Infant formulae and food products marketed for children have been increasingly supplemented with probiotics and/or prebiotics. A vast number of studies have accounted for the transit of probiotic use from alternative to more evidence-based medicine. Data support the use of certain probiotics for the adjunct treatment of acute viral gastroenteritis, and for prevention of gastrointestinal diseases. Further roles of prebiotics and probiotics are seen in the prevention of overall infectious diseases and respiratory infections. Data from well-conducted randomized-controlled trials support the therapeutic role for probiotics toward necrotizing enterocolitis in preterm infants. However, it is difficult to translate heterogeneous-based study results, which are mainly due to varying genera, strains, doses, study settings and measured outcomes, into evidence-based recommendations. This article focuses on the evidence of clinical benefits of prebiotics, probiotics and synbiotics toward prevention and treatment of pediatric infectious diseases.

Key Words: probiotics, prebiotics, pediatric infectious diseases

(*Pediatr Infect Dis J* 2012;31: 859–862)

In the last decade, infant formulae and food products marketed for children have been increasingly supplemented with probiotics, prebiotics, or with the combination of both, synbiotics. Distribution and use of such products seem to be ahead of our basic understanding of how probiotics work and of what long-term impact they have on modulation of our gut microbiota. Also, interpretation and extrapolation of data is limited, mostly due to a high heterogeneity of clinical studies with regard to varying genera, strains, doses, study settings and measured outcomes. Nevertheless, with an increasing number of well-conducted clinical studies, the body of evidence for or against the use of probiotics and prebiotics is growing. This article will focus on the evidence of clinical benefits of prebiotics, probiotics and synbiotics toward prevention and treatment of pediatric infectious diseases.

DEFINITIONS AND RATIONAL FOR USE

Probiotics are live microorganisms that, when administered in adequate amounts, confer a health benefit on the host.[1] In contrast, prebiotics are nondigestible food

immune-modulatory functions, influence and promote epithelial cell differentiation, proliferation and intestinal barrier function in vitro.[3] Commercialized prebiotics have been developed to successfully mimic the prebiotic effects of human milk oligosaccharides found in human breast milk. They are used to selectively promote colonization, growth, survival and function of commensal bacteria and possibly modulate the immune system.[4] Due to their structural differences to human milk oligosaccharides, they might lack the ability to promote further health benefits beyond their prebiotic function.[5]

PREVENTION OF OVERALL INFECTIOUS DISEASES

In a recent observational study (771 infants), the group receiving a follow-on formula supplemented with synbiotics had significantly less overall infectious diseases (ID) compared with the control group (31.0% versus 40.6%; $P < 0.05$), whereas if analyzed for specific ID, only frequency of gastrointestinal infections remained significantly different (3.5% versus 6.8%; $P = 0.03$).[6] A randomized-controlled trial (RCT) demon-

Probiotics for treating acute infectious diarrhoea (Review)

Allen SJ, Martinez EG, Gregorio GV, Dans LF

- 1966-2010 çalışmalar
- 8012 katılımcısı olan 56 çalışma
- ishal süresi 24.76 saat, n=4555, çalışma=35)
- İshal sıklığı 2. gün, n=2751, çalışma=20)

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This is a reprint of a Cochrane review, prepared and maintained by The Cochrane Collaboration and published in *The Cochrane Library* 2010, Issue 12

Journal of Pediatric Gastroenterology and Nutri
46:619–621 © 2008 by European Society for Pe
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and Nutrition/Europ
Evidence-Based
Gastroenteritis i

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§J. Hans Hoekstra, ||R
Evidence-Based G

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Vincent de Paul, Paris, France, §Depar
Children's Medical Center, Petach-Tikv
Tel Aviv University, Israel, and ¶Me

Acute gastroenteritis (AGE) is on
diseases in children, and the sec
morbidity and mortality worldwi
expected to experience AGE in th
The attack rate ranges from 0.5 to
annually in high-income countries,
2 to 3 years of life (2.5 illnesses per
5 illnesses in those attending day ca
European countries AGE is usually
still associated with a large number
and a not negligible number of dea

Europe encompasses a large nu
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rate. Clinical practice guidelines ar
help the practitioner keep up to
best practices.

A number of guidelines for the m
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to be considerable clinical variation
AGE across Europe (6). This ma

Received February 11, 2008; accepted F
Address correspondence and reprint
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Via S. Pansini 5, 80131 Napoli, Italy (e-m
Development of the guidelines was su
GlaxoSmithKline and Merck Sharp & Doh
Conflicts of interest of the working grou
end of the article.

KEY POINTS

The reader is referred to the full-length document for the complete list of recommendations and statements resulting from the systematic review of the literature (see Supplement to this issue of the *Journal*). The main conclusions and recommendations emerging from this project are listed below:

1. Acute gastroenteritis is an extremely common problem in childhood, particularly in the first 3 years of life. In Europe, it is usually, although not always, a mild disease, and death is an exceptional outcome. However, gastroenteritis is associated with a substantial number of hospitalisations and high costs.
2. The severity of gastroenteritis is related to aetiology rather than to age, and rotavirus is responsible for the most severe cases.
3. Dehydration is the main clinical feature of acute gastroenteritis and generally reflects disease severity. Weight loss, prolonged capillary refill time, skin turgor, and abnormal respiratory pattern are the best individual clinical signs of dehydration.
4. Hospitalisation should be reserved for children in need of procedures that can only be carried out in hospital, such as intravenous rehydration.
5. Microbiological investigations are generally not needed.
6. Rehydration is the key treatment and should be applied as soon as possible. Reduced osmolality oral rehydration solution should be used, and it should be offered ad libitum.
7. Regular feeding should not be interrupted and should be carried on following initial rehydration. Regular milk (lactose-containing) formulas are appropriate in the vast majority of cases.
8. Drugs are generally not necessary; however, selected probiotics may reduce the duration and intensity of symptoms. Other drugs may be effective but require further investigations.
9. Antibiotic therapy is not needed in most cases of AGE and may induce a carrier status in case of *Salmonella* infection. Antibiotic treatment is effective mainly in shigellosis and in the early stage of *Campylobacter* infection.
10. Prevention with antirotavirus vaccination is recommended for all European children and is expected to consistently reduce the burden of gastroenteritis and to prevent most of the severe cases in the most susceptible age groups.

ogy, Hepatology,
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itive Summary

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astroenteritis in

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University Paris 5, and Hôpital Saint
bosch, The Netherlands, ||Schneider
iseases, Sackler Faculty of Medicine,
ics (II Katedra Pediatrii), Poland

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widespread adoption.
European Society for Paediatric
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ciety for Paediatric Infectious
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line papers, 1 devoted to the
the otherwise healthy child with
otavirus vaccination.

THE GUIDELINES

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rs to reach decisions based on
analysis.

S DEVELOPMENT

veloped by an ESPGHAN/ESPID
ted by 7 experts from France, the
, and Poland, and coordinated by
rsity of Naples Federico II).

ARTICLE

A Meta-analysis of the Effects of Oral Zinc in the Treatment of Acute and

Marek Lukacik, MD[a], Ronald L. Thomas, PhD

[a]Department of Pediatrics, Children's Medical Ce... hool of Medicine, and Children's Hospital of Michigan, D... Research Unit Network, Wayne State University

The authors have indicated they have no financi...

- Çinko hücreleri oksidatif stresden korur
- İshalde büyük miktarda çinko kaybı olur

- 22 RKÇ (16 akut ishal (n:15231, 6 persistan ishal (n:2968)
- Plasebo ile karşılaştırıldığında ishal sıklığı ve miktarı azalıyor

- Teori: su ve elektrolit abzorbsiyonunu artırıyor, ancak kesin mekanizma bilinmiyor

ABSTRACT

OBJECTIVE. Children in develo... Supplemental zinc has prev... diarrhea. The objective of ... supplemental oral zinc the...

METHODS. We conducted a me... the efficacy and safety of supple... and persistent diarrhea. Results we... weighted mean difference. A total of ... n: 18 examined acute diarrhea (n = 15 231), an... ne... a (n = 2968).

RESULTS. Mean duration of a... rrhea and persistent diarrhea was significantly lower for zinc compared w... cebo. Presence of diarrhea between zinc and placebo at day 1 was not significantly different in acute diarrhea or persistent diarrhea trials. At ... 3, presence was significantly lower for zinc in persistent diarrhea trials (n = ... but not in acute diarrhea trials. Vomiting after therapy was significantly higher ... zinc in 11 acute diarrhea trials (n = 4438) and 4 persistent diarrhea trials (n = 2969). Those who received zinc gluconate in comparison with zinc sulfate ... etate vomited more frequently. Overall, children who received zinc reported an 18.8% and 12.5% reduction in average stool frequency, 15.0% and 15.5% shortening of diarrhea duration, and a 17.9% and 18.0% probability of reducing diarrhea over placebo in acute and persistent trials, respectively.

CONCLUSIONS. Zinc supplementation reduces the duration and severity of acute and persistent diarrhea; however, the mechanisms by which zinc exerts its antidiarrheal effect have not been fully elucidated.

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...inc

...eviations

WHO—World Health Organization
ORS—oral rehydration solution
RR—relative risk
WMD—weighted mean difference
CI—confidence interval
cAMP—3′,5′-cyclic monophosphate
K—potassium
Ca—calcium

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Viral gastroenterit korunma

- Küçük bebeklerde emzirme viruslara (özellikle rotavirusa) karşı koruma sağlar
- El yıkamak gibi iyi hijyen kuralları özellikle bakım evlerinde ve hastanelerde etkili bir korunma stratejisi
- Dezenfektanlara karşı norovirus kısmen dirençli olsada kontamine yüzeylerin temizliği önemli
- Gastroenteritde virus bulaşını istendiği biçimde azaltmak zor:
- A) düşük virus yükü ile bulaş olabilir
- B) enfekte kişinin kusmuğunda ve dışkısında yüksek viral yük
- C) ajanlar çevrede oldukça stabil

En iyi korunma yolu: AŞI

DSÖ 5 yaşın altında ishale bağlı mortalitenin %10 < olan ülkelerde rotavirus aşıını rutin olarak önermektedir

Rotavirus aşısı

- **Monovalan Human Rotavirus Aşısı (HRV, Rotarix®)**
- G1P1A[8] suşunu içeren canlı, attenüe bir aşı

- **Pentavalan Human-Bovine Reassortant Rotavirus Aşısı (PRV, Rotateq®)**
- WC-3 sığır rotavirus G6 P5 [7] ile insan VP7 G1-G4 ve VP4 P1A[8] reassortantı olan beş valanlı canlı, oral aşıdır.

Rotavirus aşısı

	PRV (RotaTeq®)	**HRV (Rotarix®)**
Doz sayısı	3	2
Önerilen şema	2- 4- 6. ay	2- 4. ay
İlk doz için minimum yaş	6 hafta	6 hafta
İlk doz için maksimum yaş	14 hafta 6 gün	14 hafta 6 gün
İki doz arasındaki minimum süre	4 hafta	4 hafta
Son doz için maksimum yaş	8 ay	8 ay

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AKUT GASTROENTERİT NEDENİYLE HASTANEYE YATAN HASTALARDA ETKENLER VE KLİNİK

ÇALIŞMA

ETIOLOGIC FACTORS AN

HOSPITALIZED CHILD

EPIDEMIOLOGIC STUD

•2008-2009 yıllları arasında ishal nedeniyle yatış %9.3

Fatih GÜ

*Ankara Ço

Tablo 3. Viral ve amibiyazisli olguların dehidratasyon decereleri

	Hafif dehidratasyon n (%)	Orta dehidratasyon n (%)	Ağır dehidratasyon n (%)
Rotavirüs	113 (%59.5)	73 (%38.4)	4 (%2.1)
Adenovirüs	9 (%75)	3 (%25)	0 (%0)
Rotavirüs + Adenovirüs	7 (%58.3)	5 (%41.7)	0 (%0)
Amibiyazis	61 (80.3)	15 (19.7)	0 (%0)

ÖZET

Akut gast deki gastroente davi fırsatı sağ . Bu çalışmanı akut gastroente aita örnekleri r ve bakteriye neye yatan has ken, %42.3'ünde etken saptanamadı. İshalli vakaların büyük çoğunluğunu erkekler oluşturuyordu. Rotavirüs en sık ilk 2 yaşta, amibiyazis ise en sık 5 yaş üzerinde tespit edildi. Rotavirüs en sık Nisan ayında (%16.8) ve ilkbahar mevsiminde (%43.7) görüldü. Amibiyazisli olgular en sık Eylül ayında (%14.5) ve yaz mevsiminde tespit edildi (%32.9). Viral etkenlere bağlı hastanede yatış süreleri daha uzun bulunurken (rotavirüs 4 gün, adenovirüs 5.7 gün, rotavirüs+adenovirüs 4.5 gün), amibiyazisli olgularda daha kısa (3 gün) bulundu.

Anahtar Sözcükler: Akut gastroenterit, çocuklar, hastaneye yatış

TEŞEKKÜRLER