his case is difficult. Meningitis in patients with inner ear malformations is associated with bacteria (e.g., *S. pneumoniae* and *Haemophilus influenzae*) that colonize the upper airways. The prevalence of oropharyngeal colonization with GBS is low (≈5%), explaining the rarity of GBS meningitis (*10*). Unlike for pneumococcal meningitis, which can be prevented at least partially by vaccination, no vaccine is available for GBS.

Our report adds another example to the growing spectrum of invasive GBS disease beyond infancy. GBS is uniformly susceptible to penicillin; therefore, treatment directed at common causes of bacterial meningitis is also appropriate for GBS (*1*,*10*). Cochlear implant recipients with symptoms of fever, otitis media, or headache should be carefully assessed; if meningitis is diagnosed, GBS should be considered as a possible causative organism.

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Severe Necrotizing Pneumonia in Children, Houston, Texas, USA

To the Editor: Routine vaccination of children with the 7-valent pneumococcal conjugate vaccine (PCV-7; Wyeth Pharmaceuticals, Collegeville, PA, USA), initiated in the United States in 2000, was followed within 2 years by an extensive and rapid decline in invasive pneumococcal disease (IPD) (*1*). During the past few years, increasing frequency of invasive disease including necrotizing pneumonia caused by serotypes not included in the vaccine has been reported (*2*). We show an expanded pattern of the changing spectrum of the disease associated with nonvaccine serotypes through this report of 4 cases of necrotizing pneumonia in children, caused by *Streptococcus pneumoniae* serotype 19A.

Over a 6-month period ending in March 2008, 4 children (median age 3.6 years, 1 with asthma) (Table) were brought to our hospital with signs of respiratory distress and a 4- to 7-day history of fever and cough. All had decreased breath sounds or crackles, and radiologic studies showed evidence of complicated pneumonia, which led to hospital admission (3 to an intensive care unit [ICU]). *S. pneumoniae* 19A was isolated from normally sterile sites with each child. All received intravenous antimicrobial drugs followed by an oral antimicrobial drug regimen and were discharged in good health. By reviewing immunization records, we confirmed that all had completed the PCV-7 series before becoming ill.

During the same period, complicated pneumonia was identified in 7 other inpatients by using the International Classification of Diseases, 9th revision, codes for necrotizing pneumonia and empyema and Current Procedural Terminology codes

Table. Characteristics of patients in study of severe necrotizing pneumonia in children, Houston, Texas, USA, 2007–2008*

Characteristic	Patient 1	Patient 2	Patient 3	Patient 4
Age, y	4.1	2.8	3.4	3.7
Race	Black	White	Black	Black
Sex	M	M	F	F
Completed PCV-7 series	Yes	Yes	Yes	Yes
Co-illnesses	Asthma	None	None	None
Clinical signs				
Date	2007 Nov	2007 Nov	2007 Dec	2008 Feb
Temperature, °C	37.6	**38.9**	37.9	**38.7**
Pulse, beats/min	**154**	**160**	**162**	**188**
Respiratory rate, breaths/min	**70**	**60**	**40**	**42**
Blood pressure, mm Hg	95/53	**122/84**	**110/78**	106/69
Oxygen saturation on room air, %	**85**	**90**	100	**86**
Symptoms/signs (hospital)	Fever, cough, dyspnea, headache, abdominal pain, decreased activity	Fever, cough, dyspnea, congestion	Fever, cough	Fever, cough, decreased appetite
Hospital course				
Site pathogen isolated	Blood and pleural fluid	Blood and pleural fluid	Pleural fluid	Blood
IV antimicrobial drugs, d	21	9	14	33
IV antimicrobial drugs (MIC, μg/mL)	Cefotaxime (0.06)	Vancomycin (0.75); ceftriaxone (0.19)	Vancomycin (1); resistant to cephalosporin	Vancomycin (0.50); ceftriaxone (0.064)
VATS† (duration, d)	Yes (2)	Yes (2)	Yes (2)	No
ICU stay	Yes	Yes	No	Yes
Intubated (duration, d)	Yes (5)	Yes (5)	No	Yes (22)
Duration of hospitalization, d	22	11	15	28
Oral antimicrobial drugs after discharge (duration, d)	Cefdinir (7)	Cefdinir (14)	Linezolid (14)	Cefdinir (7)

*PCV, pneumococcal conjugate vaccine; IV, intravenous; VATS, video-assisted thoracoscopic surgery; ICU, intensive care unit. **Boldface** indicates clinically significant differences.
†Hospitalization day that VATS was performed.

for thoracoscopic surgery (median age 4.3 years); 2 had asthma, 1 had congenital diaphragmatic hernia with resultant left lung hypoplasia. No causative organism was identified for any of these cases.

As illustrated by our 4 cases, serotype 19A is emerging as an increasing cause of severe disease such as complicated pneumonia. Although the incidence of IPD in general has decreased since the introduction of PCV-7, the emergence of nonvaccine serotypes as a cause of severe disease is becoming more prevalent. Among 8 geographic areas in the United States, the incidence of IPD caused by nonvaccine serotypes in children <5 years of age increased from 16.3 cases/100,000 population to 19.9 cases/100,000 population, respectively, from prevaccine years 1998–1999 to 2004 (*3*).

Because organisms were not isolated from the 7 other patients with necrotizing pneumonia during the same period, we are unable to comment on whether *S. pneumoniae* 19A was the predominant cause of necrotizing pneumonia in our study. However, in comparing our patients with these 7 patients, those with *S. pneumoniae* 19A appear to have had a more complicated course of illness, longer hospital stays (mean 19 days vs. 13 days), and a longer course of intravenous antimicrobial drugs (mean 19.2 days vs. 17 days). Although these 7 patients required more video-assisted thoracoscopic surgery than did our 4 patients (100% vs. 75%), those with *S. pneumoniae* 19A necrotizing pneumonia had a more severe clinical course of illness resulting in more ICU admissions (75% vs. 29%) and intubations (75% vs. 14%).

All 4 of our patients had completed the PCV-7 series before becoming ill. The emergence of nonvaccine serotypes as a cause of severe disease may be caused by serotype replacement and increased nasopharyngeal carriage of nonvaccine serotypes after receiving PCV-7 (*4*). Our report supports the theory of serotype replacement. The increasing incidence of invasive disease caused by nonvaccine serotypes has prompted development of an expanded pneumococcal vaccine to include serotypes 1, 3, 5, 6A, 7F, and 19A in addition to those covered by PCV-7 (*5*). The need for this expanded vaccine is becoming more evident as the number of children with severe pneumococcal disease due to current nonvaccine serotypes increases.

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Human Bocavirus 2 in Children, South Korea

To the Editor: In 2009, Kapoor et al. and Arthur et al. published reports on the prevalence of the newly identified parvovirus, human bocavirus 2 (HBoV-2), in fecal samples (*1*,*2*). HBoV-1 had been discovered in 2005 (*3*), and reports indicate its possible role in respiratory diseases such as upper respiratory tract infections, lower respiratory tract infections (LRTIs), and in exacerbation of asthma (*4*); in these diseases, the virus co-infects with other respiratory viruses (*5*). Systemic infection with HBoV-1 and possible association of this virus with other diseases such as gastroenteritis, Kawasaki disease, and hepatitis have been reported (*6*–*8*). We looked for HBoV-2 in clinical samples from children with various diseases, including acute LRTIs, Kawasaki disease, Henoch-Schönlein purpura, and hepatitis.

During September 2008–January 2009, a total of 212 nasopharyngeal aspirates were collected from 212 children (median age 8 months, range 1–59 months) hospitalized with acute LRTIs at Sanggyepaik Hospital in Seoul, South Korea. Previously, during January 2002–June 2006, a total of 173 serum samples had been obtained from children (age range 1 month–15 years) with hepatitis (hepatitis B, 20 samples; hepatitis C, 11 samples; unknown hepatitis, 31 samples), Kawasaki disease (12 samples), and Henoch-Schönlein purpura (18 samples) and from healthy children (same age range, 81 samples) (*9*). The study was approved by the internal review board of Sanggyepaik Hospital.

DNA was extracted from serum samples, and RNA and DNA were extracted from nasopharyngeal aspirates by using a QIAamp Viral RNA Mini Kit (QIAGEN, Hilden, Germany) and a QIAamp DNA Blood Mini Kit (QIAGEN GmbH), respectively. All nasopharyngeal aspirates were tested by PCR for common respiratory viruses such as respiratory syncytial virus, influenza viruses A and B, parainfluenza virus, and adenovirus, as described previously (*10*). PCRs to detect HBoV-1 were performed by using primers for the nonstructural (NS) 1 and nucleocapsid protein (NP) 1 genes, as described previously (*10*). Additional PCRs for rhinovirus, human metapneumovirus, human coronavirus (hCoV)-NL63, hCoV-OC43, hCoV-229E, hCoV HKU-1, WU polyomavirus, and KU polyomavirus were performed, as described, for HBoV-2 –positive samples (*10*).

HBoV-2 was detected by performing first-round PCR with primers based on the *NS* gene, HBoV2-sf1, and HBoV2-sr1. Second-round PCR was performed by using primers HBoV2-sf2 and HBoVsr2, as described previously (*1*). The PCR products were sequenced by using an ABI 3730 XL autoanalyzer (Applied Biosystems, Foster City, CA, USA). The nucleotide sequences were aligned by using BioEdit 7.0 (www.mbio.ncsu.edu/BioEdit/BioEdit.html) and presented in a topology tree, prepared by using MEGA 4.1 (www.megasoftware.net).

Of the 212 samples tested, the following viruses were detected: human respiratory syncytial virus (in 124 [58.4%] samples), human rhinovirus (24 [11.3%]), influenza virus A (18 [8.4%]), adenovirus (10 [4.7%]), and parainfluenza virus (8 [3.7%]). HBoV-1 was not detected in the study population. HBoV-2 DNA was found in 5 (2.3%) of the 212 samples collected; all positive samples had been obtained in October 2008. The age range of the children with HBoV-2–positive samples was 4–34 months (median 24 months), and all were male. The diagnoses were bronchiolitis for 3 children and bronchopneumonia for 2. The most frequently codetected virus was human respiratory syncytial virus, found in 4 (80%) of 5 samples. One