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Recognizing Septic Shock in Young Infants: An Illustrative Case from the King County Child Death Review Team

Special thanks to Brian Johnston, MD MPH, Chief of Pediatrics at Harborview Medical Center for contributing this article.

The following is a composite case which combines details from more than one case to preserve confidentiality. It illustrates an issue in pediatric mortality identified by the multi-disciplinary King County Child Death Review Team:

A 3-month-old previously healthy infant was taken to a local emergency department for evaluation of fever. The child had mild upper respiratory symptoms without cough. He had no vomiting or diarrhea. There were no ill contacts. Fever had developed 2 hours before presentation.

On examination, the child was described as irritable, but "non-toxic" in appearance. He had a temperature of 40°C, a respiratory rate of 30, and a pulse of 220. There were no focal findings on physical exam. There was no rash. His extremities were described as cool. WBC was 8,000 with no band forms. Urinalysis was remarkable only for a specific gravity of 1.030. A blood culture was obtained, and an IV placed. To address poor perfusion, the child received 40 cc/kg of isotonic fluids over a 3 hour period. His heart rate was 180 at discharge. The parents were instructed to follow-up with their primary care provider the next day.

Four hours after ED discharge the parents noted that the child's breathing was labored. He had developed a petechial rash. The parents then noted seizure-like activity during which the infant became apneic. The family initiated CPR, and called for assistance. The child died at a tertiary referral center 2 hours later. The blood culture, drawn less than 12 hours earlier in the ED, grew *Neisseria meningitidis*, serotype B.

This infant presented in septic shock and died from fulminant meningococcemia. Infants and children with febrile illness account for up to 20% of visits to a general Emergency Department. Most have benign, self-limited illnesses and require only supportive care and parental reassurance. The challenge remains to identify the small proportion of febrile children with invasive bacterial disease requiring aggressive therapy. Unfortunately, as this case illustrates, early signs of septic shock in infants may be subtle while the progression of the disease is rapid and dramatic.

Sepsis is a clinical diagnosis and does not rely upon isolation of a specific organism¹. Septic shock should be suspected in an infant with evidence of an infection (manifest as fever or hypothermia) who also has clinical signs of decreased perfusion, including altered mental status (irritability, lethargy, lack of interaction with parents), decreased urine output, peripheral vasodilation ("warm" shock) or vasoconstriction ("cool" shock). Hypotension is a late finding and is not required for the clinical diagnosis of septic shock.

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The American College of Critical Care Medicine has developed treatment guidelines for the hemodynamic support of pediatric and neonatal septic shock². Early and aggressive fluid therapy is recommended to restore circulating volume in the first hour of therapy. Large deficits typically exist, and initial volume resuscitation may require up to 60 cc/kg over 15 minutes. Studies from community samples suggest that each hour of delay in reversing shock *doubles* the odds of death for the child³.

Protein-conjugate vaccines have dramatically reduced the incidence of invasive *Haemophilus influenzae* type b^4 and, more recently, Streptococcus pneumoniae among infants and young children⁵. With these diseases on the wane, practitioners may be tempted to abandon previously published guidelines that called for careful laboratory evaluation and empiric treatment of febrile infants. Nevertheless, serious bacterial disease remains a concern in this age group and a high level of clinical suspicion is warranted. Organisms still capable of causing invasive disease in infants include : Haemophilus influenzae type b or pneumococcus in the un-immunized, partially immunized or fully immunized child (due to vaccine failure); non-type b H. flu strains, pneumococcal strains not covered by the heptavalent conjugate vaccine; late-presenting group B streptococci; Neisseria meningitidis; Salmonella species; and other Gram-negative enteric bacilli. Risk factors for occult bacterial infection in the febrile infant include toxic appearance, altered mental status, higher fever, and younger age.

Although still an uncommon disease, invasive meningococcal infection is has its highest incidence among infants⁶ and early recognition and treatment remains the mainstay of control⁷. Opportunities to prevent disease through vaccination in infancy are limited because more than half of cases among those less than 1 year of age are caused by serogroup B, for which no vaccine is licensed or available in the United States. For other serotypes, the currently available meningococcal polysaccharide vaccines provide limited efficacy of short duration in young children^{7.} Protein conjugate serogroup C meningococcal immunization was recently introduced in the United Kingdom and may augment our ability to control a proportion of meningococcal disease among children when this vaccine becomes available in the United States⁸.

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Breaking Out All Over: Norovirus

This fall and winter, Public Health has received several reports of viral gastroenteritis outbreaks at health care facilities, long-term care facilities, schools, and athletic events. Several of these outbreaks have been confirmed by the Washington Public Health Laboratory as being caused by norovirus. In November 2003, Public Health received several reports of a norovirus-like disease in people who consumed raw oysters.

The term norovirus was first introduced in the February 2003 issue of the *Epilog* as the new name for the group of viruses previously called Norwalk-like viruses from the family *Caliciviridae*. These viruses cause acute gastroenteritis in humans characterized by nausea, vomiting, diarrhea, and abdominal cramps. The incubation period for norovirus ranges from 24 to 60 hours: symptoms typically start abruptly, and usually last only one to two days.

The virus is present in the feces and vomitus of an infected person, and viral shedding can occur for prolonged periods, even in the absence of clinical disease. With an infective dose of fewer than 100 viral particles, the disease spreads easily from person-to-person, and through contaminated food or water. Though fecal contamination can render any food a source for norovirus transmission, raw or undercooked oysters, and clams harvested from feces-contaminated water are often implicated.

Clusters of norovirus-like illness, such as those in healthcare facilities, residential settings, or long term care facilities, and cases thought to be caused by contaminated food or water, should be reported to Public Health by calling (206) 296-4774. Laboratory testing for noroviruses is not currently available at most commercial laboratories. However, in outbreak situations, with prior approval through Public Health-Seattle & King County, testing of feces and/or vomitus for norovirus is available at the Washington State Public Health Laboratory. For more information about norovirus infection, including infection control measures, go to:

http://www.metrokc.gov/health/epilog/vol4302.htm#norovirus http://www.cdc.gov/ncidod/dvrd/revb/gastro/norovirus.htm

Update on H5N1 Avian Influenza and SARS H5N1 Avian Influenza

The large-scale avian flu outbreak among birds is continuing in Asia. To-date, 34 cases (23 fatal) of human infection due to avian influenza H5N1 have been reported to the WHO; 22 from Vietnam, and 12 from Thailand. There are still no confirmed cases of human-to-human transmission of the virus, and the outbreaks have not spread to other countries.

SARS

There is currently no evidence of person-to-person transmission of SARS in the world. Four cases of SARS have been reported from China since mid-December 2003. None of these cases resulted in spread to other contacts

Current clinical guidelines for avian influenza and SARS are available at:

http://www.metrokc.gov/health/sars/sarsadvisory040203.htm

Disease Reporting

AIDS/HIV	. (206) 296-4645			
STDs	. (206) 731-3954			
TB (206) 731-4579				
All Other Notifiable Communicable Diseases (24 hours a day)	. (206) 296-4774			
Automated reporting line for conditions not immediately	(000) 000 4700			
notifiable	. (206) 296-4782			
<u>Hotlines</u>				
Communicable Disease				
HIV/STD	. (206) 205-STDS			
Online Resources				
Public Health Home Page: <u>www.metrokc.gov/health/</u>				

The *EPI-LOG*: <u>www.metrokc.gov/health/providers</u> Subscribe to the Public Health Communicable Disease listserv (PHSKC INFO-X) at: <u>http://mailman.u.washington.edu/mailman/listinfo/phskc-info-x</u>

Reported Cases of Selected Diseases	s, Seattle &	King Cou	nty 2004	
	Cases Reported		Cases Reported	
	in February		Through February	
	2004	2003	2004	2003
Campylobacteriosis	14	18	31	34
Cryptosporidiosis	2	2	3	4
Chlamydial infections	402	328	790	696
Enterohemorrhagic E. coli (non-O157)	0	0	0	0
E. coli O157: H7	0	2	0	6
Giardiasis	10	4	23	17
Gonorrhea	98	96	210	225
Haemophilus influenzae (cases <6 years of age)	0	0	0	0
Hepatitis A	2	1	2	3
Hepatitis B (acute)	5	2	7	5
Hepatitis B (chronic)	53	48	87	102
Hepatitis C (acute)	0	2	0	2
Hepatitis C (chronic, confirmed/probable)	62	106	173	225
Hepatitis C (chronic, possible)	22	25	56	50
Herpes, genital (primary)	60	52	114	112
HIV and AIDS (includes only AIDS cases not previously reported as HIV)	50	51	68	84
Measles	0	0	0	0
Meningococcal Disease	1	0	5	1
Mumps	0	0	0	0
Pertussis	21	10	44	35
Rubella	0	0	0	0
Rubella, congenital	0	0	0	0
Salmonellosis	7	11	24	35
Shigellosis	3	8	19	16
Syphilis	6	5	11	13
Syphilis, congenital	0	0	0	0
Syphilis, late	11	6	15	8
Tuberculosis	8	14	16	24

The *Epi-Log* is available in alternate formats upon request.