EGNR Bacteremia Complicates Rotavirus Disease

BY CHRISTINE KILGORE
Contributing Writer

Children with rotavirus gastroenteritis face a small but real risk of developing enteric gram-negative sepsis, investigators have reported.

Physicians "should be aware of the possibility of this complication, especially when a child is developing a high fever and lethargy several days after the beginning of gastroenteritis," said Amos Adler, M.D., and his colleagues at the Sapir Medical Center in Kfar Saba, Israel.

"In such cases, prompt initiation of wide-spectrum antibiotics is crucial, even in previously diagnosed rotavirus infection," they said (Clinical Pediatrics 2005:44;351-4).

The investigators described three previously healthy infants who developed enteric gram-negative rods (EGNR) bacteremia during rotavirus gastroenteritis. The children were hospitalized at the medical center in 2000 and 2001.

The infants had the characteristic clinical course of rotavirus gastroenteritis at the beginning of their illness. Then, 3-5 days after the onset of disease, they presented with an abrupt onset of high fever, lethargy, and poor perfusion.

Laboratory results suggested bacterial sepsis, and in one case, there also was radiographic evidence of severe intestinal injury due to pneumatosis intestinalis.

In all of them, the EGNR isolated from the blood cultures were sensitive to aminoglycosides and to second- or third-generation cephalosporins.

(Stool cultures in each patient tested negative for *Shigella, Salmonella*, enteropathogenic *Escherichia coli*, and *Campylobacter*.)

Differentiating between secondary EGHR infection and the deterioration of rotavirus gastroenteritis to a severe course "may be difficult," the investigators said.

It also is difficult to pinpoint the mechanism of bacterial breakthrough and spreading in these three cases, especially since rotavirus is not known to cause extensive inflammation and cell destruction, they said.

Still, the investigators said, they hypothesize that the pathogenesis of the bacteremia "was dissemination of normal intestinal flora through the damaged mucosa"— just as viral infection of the respiratory tract can antecede and predispose children to colonization and invasion of bacteria such as *Streptococcus pneumoniae.*

It is possible that bacteremia took hold through other sites—the urinary tract or the respiratory tract, for instance—but it's less likely since no clinical or laboratory findings support it, Dr. Adler and his colleagues said.

They said they could not find in the English literature a description of EGNR bacteremia as a complication of rotavirus infection.

One of their patients, for example, was a healthy 9-month-old boy, admitted after 1 day of vomiting and diarrhea. On admission, he was afebrile and appeared lethargic and moderately dehydrated.

The child had normal blood count and electrolytes, urea 53 mg/dL, mild metabolic acidosis and normal urine analysis. His general condition improved after treatment with intravenous fluids. His diarrhea continued, but vomiting subsided. Stool bacterial cultures were negative, and rotavirus antigen was detected in his stool.

On the third day of hospitalization, his temperature rose to 39.5° C, and he became more lethargic.

A plain abdominal radiograph showed intraluminal air in the small bowel (pneumatosis intestinalis) without free air or intraportal gas. Abdominal ultrasound appeared normal. Laboratory analysis showed white blood cell counts of 14,400 cells/μL with 9% bands and 59% neutrophils, urea 15 mg/dL, pH 7.37, partial pressure of carbon dioxide 20.7 mm Hg, bicarbonate 11.8 mmol/L, and normal U/A.

The infant was treated with intravenous piperacillin-tazobactam, and oral feeding was discontinued. His fever resolved, and his general condition improved. *Klebsiella pneumoniae* was recovered from the blood culture and was sensitive to cephalosporins, aminoglycosides, trimethoprim-sulfamethoxazole, and amoxicillin-clavulanate.

After 4 days of fasting, the infant began receiving semi-elemental nutrition. The infant completed 10 days of intravenous antibiotics and resumed a normal diet by the 13th day of hospitalization. He was discharged and appeared to be in excellent health at follow-up 1 month later. ■