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Worms and Other Stuff in the Mouth

SPECIAL REPORT

"In Mexico we have a word for sushi: Bait." —Jose Simon

Early on a recent saturday morning, a 49-year-old male called because, while sitting comfortably reading his morning newspaper, he found a live, wriggling worm in his mouth. He felt otherwise well, but did recall that two nights earlier, he had difficulty getting to sleep because of a sensation that something was caught in the back of his throat. He denied abdominal pain, vomiting, or diarrhea, but indicated he took lansoprazole to treat gastroesophageal reflux disease.

He had no pets and had no relevant travel history. He had no unusual dietary habits, but did often eat sushi and, in addition, ate salmon at home several days prior to his phone call; he believed the salmon had been well cooked.

Upon examination, the worm was a third-stage larva of *Anisakis simplex*, a "sushi worm."

COMMENT BY STAN DERESINSKI, MD, FACP

Although affected patients somehow miss the humor, nothing elicits attempts at jocularity by medical authors like the sushi worm, the cause of anisakiasis. (*See Table 1.*) Human anisakiasis (also called anisakidosis) is the result of the accidental ingestion of the larvae of one of several anisakid nematodes. (*See Table 2.*) *A. simplex* and, with lesser frequency, *Pseudoterranova decipiens* have been responsible for all reported North American cases.[1-4] All the etiologic agents of anisakiasis belong to the family Anisakidae of the superfamily Ascaroidea.[5]

The infection may be invasive or noninvasive and symptomatic cases of anisakiasis may present in one of several ways. As the result of the occasional cephalad migration of the larval nematode from the stomach, it may be discovered as a consequence of its causing a tickling sensation in the oronasopharynx ("tingling throat syndrome"). The worm has also been discovered extruding from the nares of a

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child and it may also be expelled as the result of coughing or vomiting. Each of these presentations may occur from one day to two weeks after ingestion of the parasite. An anisakid worm has also been found in the resected tonsil of a child with chronic recurrent tonsillitis.[6]

Having found the stomach a salubrious environment, the parasite may attempt to burrow into the gastric mucosa, with resultant acute abdominal pain, vomiting, and hematemesis occurring within less than an hour of ingestion of the worm.[7-9] Leukocytosis is common but significant eosinophilia is not. Patients may present with severe chest pain that must be distinguished from cardiac ischemia.[10,11] Although chest pain is usually due to gastric involvement, the parasite may also burrow into the esophageal mucosa.[12] Anisakiasis may also be associated with a "vanishing tumor" of the stomach or, less frequently, other sites in the gastrointestinal tract.[5] Therapy of gastric (or esophageal) anisakiasis consists of endoscopic extraction of the parasite. Endoscopic examination of 87 patients with gastric anisakiasis found that 55% of the worms were found in the greater curvature.[13]

Table 1

Some Notable Titles of Medical Articles About Sushi

"If you knew sushi like I know sushi"[53]
"Sushi syncope schusser gets wedeler's valve at Vail"[54]
"Hot tubs, sex, sushi, and infectious diseases"[55]
"Anisakiasis: Revenge of the sushi parasite"[56]
"Hold the sushi"[57]
"Horseradish horrors: Sushi syncope"[58]

Organisms that make their way farther along the gastrointestinal tract may burrow into the intestinal wall to the level of the muscularis mucosae and, occasionally, may penetrate transmurally and find their way to the peritoneal cavity. Worms that have penetrated tissues elicit a granulomatous response and die, not necessarily in that order. This may result in intestinal obstruction, an omental mass lesion, fever, and pain.[14-16] Intestinal symptoms usually begin within 48 hours of ingestion of the parasite and may last one to five days. Intestinal obstruction is associated with segmental involvement of the small bowel.[17] Intestinal anisakiasis or involvement of mesenteric lymph nodes may each cause findings that can be confused with acute appendicitis.[16,18] *A. simplex* has caused peritonitis in a patient undergoing continuous ambulatory peritoneal dialysis.[19] Anisakids that are not ejected anally and do not penetrate tissue are eliminated spontaneously within three weeks of infection.

In addition to these direct effects of the parasite, reports from Spain strongly suggest that, despite the usual absence of significant eosinophilia, anasakid worms may be responsible for a variety of allergic manifestations, including urticaria and anaphylaxis.[20] A study of 26 patients who had urticaria or angioedema 20 minutes to 23 hours after eating raw or undercooked fish, only one-half of whom had abdominal symptoms, found a high frequency of IgE antibody and positive skin prick tests to *A. simplex* antigens, relative to controls.[21] Serologic studies have demonstrated an association between sensitization to *A. simplex* and some cases of eosinophilic gastroenteritis.[22]

Patients with allergic manifestations after eating infected fish may subsequently ingest the same species of fish without elicitation of symptoms, if the fish are not infected.[23] On the other hand, allergic reactions may subsequently occur as the result of ingestion of fish containing nonviable *A. simplex*.

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Table 2

Anisakid Nematodes Implicated in Human Infections[1]

Anisakis simplex (herring worm)*
Pseudoterranova (*Phocanema*, *Terranova*) *decipiens* (cod or seal worm)*
Contracaecum spp.
Hysterothylacium (*Thynnascaris*) spp.

*Reported from North American cases

Epidemiology

Human infection by anisakid nematodes is the consequence of the ingestion of infested, raw, undercooked, or insufficiently cured fish and shellfish. The yearly number of identified and reported cases in the United States is fewer than 10, with Hawaii and California contributing the most cases. In contrast, more than 1000 cases are reported annually in Japan.[1]

Sea mammals become infected with third-stage larvae by ingesting fish or squid containing the parasite. *A. simplex* predominantly affects dolphins, porpoises, and whales, while *P. decipiens* affects seals, fur seals, walruses, and sea lions. The anisakid matures to the adult stage in the stomach of the sea mammal and eggs are subsequently excreted in the feces. These are eaten by small crustaceans, such as krill, and start their way up the food chain once again. Larvae of *A. simplex* are found predominantly in herring, salmon, cod, mackerel, and squid, while *P. decipiens*, in addition to infesting cod, are also found in halibut, flatfish, greenling, pike, bonito, Alaskan pollack, and red snapper (Pacific red snapper is actually a rockfish). Humans become infected by ingestion of fish containing viable larvae.

Diagnosis

The diagnosis of anisakiais may be made by examination of an expelled or extracted worm.[24] The third-stage larvae of *A. simplex* are 19-36 mm in length and up to 0.1 cm in diameter. They are white or milky in color and have a long stomach and intestine with absent cecum, and a blunt tail. The anterior portion of the head contains three bilobed lips, and a small boring tooth just dorsal to an excretory pore; near the rectum are three anal glands. The third-stage larvae of *P. decipiens*, which are yellow or brownish as the result of hematophagy, are 25-50 mm by 0.3-1.2 mm. They have an anteriorly projected cecum.

The diagnosis of gastric anisakiasis is best made at endoscopy, a procedure that allows definitive therapy, i.e., extraction of the parasite. If radiographic examination is, however, performed, it may reveal mucosal edema, pseudotumor formation, and a threadlike filling defect approximately 30 mm in length representing the worm.[25]

Abdominal ultrasound examination of patients with small intestinal anisakiasis often reveals ascites, small bowel dilatation, and focal edema of Kerckring's folds.[26] Double-contrast radiographic examination of patients with colonic anisakiasis may reveal right-sided luminal narrowing and thumbprinting; due to mucosal edema the worm may also be visualized.[27] Colonic anisakiasis may simulate carcinoma.[28] The worm may be extracted during colonoscopy.[29]

Histologic examination of tissue removed at surgery reveals an eosinophilic granuloma with a larva with Y-shaped lateral chords without the lateral alae (seen in Ascaris) that has 60-90 muscle cells per quadrant and 60-80 cells in the intestine.[1,25] The cuticle has three layers. *Pseudoterranova* spp. have butterfly-shaped lateral chords, and their intestine has a cecum and contains more than 100 intestinal cells.[1]

While serological testing has been used for diagnosis, particularly in patients with allergic manifestations, it is not standardized and its accuracy remains undemonstrated. There is a high degree of antigenic cross-reactivity between *A. simplex* and *Ascaris suum* and *Toxocara canis*.[30]

Pathophysiology

Anisakid infection appears to usually be caused by a single worm; however, as many as 56 have been endoscopically extracted from the stomach of a single individual.[31] Its ability to burrow through mucosa may be, at least in part, related to its enzymatic production. *A. simplex* secretes a hyaluronidase with a molecular weight of 40,000 and a pH optimum of 4.0 with no activity at neutral pH.[32] It also secretes a metallo-aminopeptidase and a tryspinlike serine protease.[1]

Infection does not appear to produce protective immunity since repeat episodes may occur.[13]

Prevention

Since infection of edible fish is common and since the worm may be difficult, if not impossible, to detect (candling is only approximately 70% sensitive in the identification of nematodes in the flesh of fish), the only means of prevention is the avoidance of inadequately prepared fish and shellfish. The FDA states that all fish and shellfish intended for raw or semi-raw (e.g., marinated, partly cooked) consumption should be blast frozen to -35°C (-31°F) or less for 15 hours, or routinely frozen to -20°C (-4°F) or less for seven days.[2] Cooking to an internal temperature of 60°C (140°F) for 10 minutes is believed to kill all nematodes and tapeworms. In addition, hard salt curing before pickling is reported to be effective. *Anisakis* larvae can survive for one day in either soy or Worcestershire sauce, six days

in 10% formalin, 51 days in vinegar, and 112 days in 1% HCL.[33] Survival is impaired in sake (16% alcohol), but not in wasabe.[5]

Other Considerations

Infection with other parasites may mimic anisakiasis. Perhaps the nematode most commonly observed in vomitus is *Ascaris lumbricoides*, but this organism is readily distinguishable by its size (10- 31 cm length in males and 22-35 cm in females). Human infection with the gullet nematode, *Gongylonema* spp. (e.g., *G. pulchrum*), results from the inadvertent entry of dung beetles or cockroaches into the oral cavity or their actual ingestion.[34,35] While, at 35 mm in the female, this parasite is similar in length to that of *A. simplex*, it is usually found imbedded within the mucosa or submucosa of the buccal cavity, tongue, or upper esophagus, presenting as an inflammatory mass.

Mammomonogamus laryngeus infection (syngamosis), almost always acquired in the Caribbean islands or in Brazil (infections have also been acquired in Korea, Thailand, and the Philippines), may mimic the "tingling throat" presentation of anisakiasis. The parasite usually infests the respiratory tracts of domestic animals, but may accidentally infect humans. Complaints have included nonproductive cough, a "lump in the throat," a crawling sensation in the throat, hemoptysis, and pneumonitis.[36,37]

A Korean patient with community-acquired pneumonia was found, on bronschoscopy, to have syngamosis; he recovered after extraction of the parasite and administration of albendazole.[15] Thiabendazole and mebendazole have also been used. The male and female of this nematode exist together in copula and can be recognized by the distinctive "Y" shape caused by this union.[36] The male is 3-5 mm and the female 9-24 mm in length. Most often, only a single pair is present. The worms are pigmented as a result of hemophagia.

Linguatula serrata, a wormlike blood-sucking parasitic arthropod of the order Pentastomida, the larval form of which is usually found in the nose, frontal sinuses, and tympanic cavity of carnivorous mammals, may cause nasopharyngeal or visceral infection in humans.[38] Nasopharyngeal linguatuliasis, also called the Halzoun-Marrara syndrome, results from the ingestion of contaminated raw or undercooked flesh of the herbivorous intermediate host, commonly sheep or goats, and represents a local hypersensitivity reaction to *L. serrata* nymphs. It may cause sore or itchy throat, congestion, and pharyngeal edema.[39] It may also result in unilateral conductive deafness, tinnitus, and facial palsy and may be complicated by otitis media.[40] A typical presentation, however, was that of a German tourist in Tunisia who developed cough, hoarseness, dysphagia, anosmia, frontal headache, and epistaxis two hours after ingestion of undercooked meat.[41] The diagnosis can be confirmed by detection of the nymphs in nasal discharge. The larval form may migrate and produce disease at other sites, including the eye.[42]

Table 3

Worms and Other Stuff in the Mouth

Anisakids
Ascaris lumbricoides (roundworm)
Syngamus laryngeus (gapeworm)
Gongylonema pulchrum (gullet worm)
Myiasis
Midge larva
Linguatula serrata (tongueworm)
Squid sperm bulbs

Other causes of wormlike forms in the mouth include oropharyngeal myiasis and infection by the larval form of the chironomid midge.[43-45] Each of these is readily recognized as being distinct from anisakids as a result of their size and segmentation; the midge larva reported by Lutwick and colleagues had three thoracic and nine elongated abdominal segments and actively swam with a serpentine motion.

The ingestion of raw squid has been associated with the immediate development of pain and burning in the oral cavity as the result of ejection from numerous spermatophores of sperm bulbs that embed themselves into the mucous membrane. These sperm bulbs grossly appear as 2-3 mm long needlelike structures. The approximately 4 cm long club-shaped spermatophores may be found in the stomach, where they may attach themselves to the gastric mucosa.[46]

A number of parasitic infestations may mimic the abdominal presentation of anisakiasis. The intestinal form of anisakiasis may be mimicked by infection with *Eustrongylides ignotus*, which has resulted from ingestion of contaminated sushi or raw minnows.[47-49] *Eustrongylides* infection has been mistaken for acute appendicitis and may cause intestinal perforation.[49] The larva is only 80-120 mm by 1-2 mm.

Gnathostomiasis may also cause intestinal lesions; *G. doloresi* has caused an obstructing colonic mass requiring resection in a man who ate raw snake meat in Japan.[50] *Angiostrongylus costaricensis*, which has occurred after the ingestion of ceviche with contaminated mint, may cause abdominal pathology, including eosinophilic ileitis with perforation.[51,52]

Enterobius vermicularis may commonly be found in appendices, but its pathologic role in causing appendicitis is uncertain. ❖

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Hepatitis A: Foodborne Outbreaks in Michigan and Maine—Should we Recommend Immunization?

ABSTRACTS & COMMENTARY

Synopsis: *Two hundred thirteen cases of hepatitis A in schoolchildren in Michigan were shown by hepatitis A genetic analysis to be transmitted by specific batches of frozen strawberries grown in Mexico and processed in California. These were widely distributed for school lunch programs. Apparently, sporadic cases in Maine and other states were subsequently linked to the same source.*

Sources: Hutin YJ, et al. *N Engl J Med* 1999;340:595-602; Koff RS. *N Engl J Med* 1999;340:644-645.

Hutin and associates on the national hepatitis A investigation team of the Centers for Disease

Control and Prevention (CDC) investigated a large outbreak of cases of hepatitis A that occurred in February and March 1997. The cases occurred almost exclusively in schoolchildren and school employees in two counties in Michigan. There had been no cases of hepatitis A reported in these counties in the preceding year. Information was obtained concerning how often a subject ate school lunch and which food items were eaten during seven school days, beginning 32 days before the peak incidence of the disease. The same information was obtained from an equal number of randomly selected classmates who did not develop hepatitis A. During the same period, 39 cases were reported from Maine and similar dietary information was obtained. In both Michigan and Maine, there was a strong association between consumption of strawberry shortcake and the subsequent development of hepatitis A. Polymerase chain reaction analyses of viruses isolated from patients in Michigan and Maine revealed an identical sequence of RNA indicating that the same virus caused the disease. It was possible to trace the strawberries responsible for the Michigan outbreak. The same RNA sequences were also found in small numbers of cases that occurred in the same general period in several other states that had received the same batches of frozen strawberries. These berries had been grown in Mexico, processed and frozen in California, and distributed though the Department of Agriculture for school lunch programs. The source of contamination was found to be probably related to unsanitary field conditions in Mexico.

In an accompanying editorial, Koff points out the increasing vulnerability of individuals in the United States to hepatitis A infections because of a falling rate (30%) of naturally acquired immunity and the increasing importation of vegetables and fruits from less developed countries where hepatitis A is endemic. The Advisory Committee on Immunization Practices (ACIP) of the CDC has recommended hepatitis A immunizations for some high-risk groups, including persons traveling or working in underdeveloped countries where hepatitis A is endemic, homosexual males, patients with chronic liver disease, and Native and Alaskan Americans. However, universal immunization has not yet been advocated. Koff believes that the time is appropriate to institute universal hepatitis A immunization in U.S. children.

COMMENT BY HAL B. JENSON, MD, FAAP

Although we have two highly effective and safe inactivated hepatitis A vaccines, they are not licensed for use in children younger than 2 years of age because of reduced efficacy. This age limit and the cost of yet another childhood vaccine have dampened enthusiasm for universal hepatitis A immunization. Hepatitis A infection most frequently occurs as sporadic cases but also occurs in the clusters and outbreaks such as these in Michigan and Maine described by Hutin et al that continue to occur and place a strain on public health departments. Physicians must provide postexposure immunoglobulin prophylaxis, and parents must take time from home and work to obtain medical care following exposure. These cases exemplify the dilemma we face to determine whether the currently available vaccines are appropriate for universal use.

In his accompanying editorial, Koff points out the increasing vulnerability of individuals in the United States to hepatitis A infections because of a falling rate (< 30%) of naturally acquired immunity, higher susceptibility of younger persons, and the increasing importa-

tion of vegetables and fruits from developing countries where hepatitis A is endemic and sanitation may be suboptimal. The ACIP has recommended routine hepatitis A immunization for persons traveling to or working in underdeveloped countries where hepatitis A is endemic, homosexual and bisexual males, persons with chronic liver disease, recipients of clotting factors, users of illicit drugs, as well as for children in high-risk communities such as Native and Alaskan Americans and children in selected areas of high hepatitis A endemicity. This approach has resulted in different hepatitis A vaccination recommendations in different areas. The state of Oklahoma recently enacted universal childhood hepatitis A immunization statewide for all children, and in the next year, Texas is implementing hepatitis A immunization for 32 counties along the Texas-Mexico border.

However, universal immunization for the entire country has not yet been recommended. Koff believes that the time is appropriate to institute universal hepatitis A immunization in U.S. children. These outbreaks demonstrate our vulnerability to global health problems despite never even leaving the confines of middle America. More and more of us, including children, are international travelers and would benefit from routine immunization in childhood.

We face barriers with hepatitis A vaccination that we have not let impede us with other vaccines. We instituted *Haemophilus influenzae* type b vaccination with the original polyribosyl ribitol phosphate (PRP) vaccine while the conjugate vaccines were being developed, to at least provide protection for all children 2 years of age and older. While combined hepatitis A and B vaccination beginning in infancy may be possible, the available hepatitis A vaccines are effective in children 2 years of age and older. We implemented universal hepatitis B vaccination in childhood even though hepatitis B, like hepatitis A, is predominantly a disease of adults and, therefore, the full public health benefit may take years or decades to realize. It is certainly appropriate to vaccinate early in life, against both hepatitis B and hepatitis A, to provide maximum protection. We have instituted universal vaccination against varicella, which, like hepatitis A, has a high rate of complete recovery but is associated with approximately 80-100 deaths annually in the United States.

We have the means by implementing universal hepatitis A vaccination to prevent much disease and even some deaths each year. However, it remains to be determined if we have the will and the determination. (*Dr. Jenson is Chief, Pediatric Infectious Diseases, University of Texas Health Sciences Center, San Antonio, TX.*) ❖

CME Questions

10. Which of the following statements is true regarding hepatitis A infections?

a. They can be reliably prevented in children younger than 2 years of age with current vaccines.
b. Universal vaccination is currently advised in areas of high epidemicity for hepatitis A.
c. Specific viral strains responsible for local outbreaks cannot be identified with tests currently available.
d. They have a high rate of acute and long-term mortality.

11. All cases of anisakiasis identifed to date in North America have been due to:

a. *A. simplex* or *Linguatela serrata.*
b. *A. simplex* or *Gongylonema pulchrum.*
c. *Ascaris lumbricoides* or *P. decipiens.*
d. *A. simplex* or *P. decipiens.*

12. Which of the following is correct?

a. The preferred treatment for gastric anisakiasis is albendazole.
b. The symptoms of gastric anisakiasis usually ensue within an hour of ingestion of infested seafood.
c. Massive eosinophilia is present in most patients with gastric anisakiasis.
d. The dominant symptom of patients with gastric anisakiasis is belching; pain is rare.

13. Which of the following is correct?

a. Candling (transillumination) has a sensitivity in the detection of nematodes in the flesh of fish of only approximately 70%.
b. The diagnosis of human anisakiasis may be made by the detection of *A. simplex* or *P. decipiens* ova in the stool.
c. Serological tests for anisakiasis are highly accurate.
d. Human infection by *A. simplex* may persist for years.

14. Which states have enacted their own hepatitis A immunization requirements?

a. Alaska, Oklahoma, and Texas
b. Oklahoma and Louisiana
c. Texas and Oklahoma
d. Texas and California

In Future Issues:

Who Put Salmonella in my Sand?

UPDATES

By Carol A. Kemper, MD, FACP

Thimerosal-containing Vaccines a Risk?

Source: A Joint Statement of the American Academy of Pediatrics (AAP) and the United States Public Health Service (PHS), July 7, 1999.

The aap and phs are recommending that all thimerosal-containing vaccines, including recombinant vaccines such as hepatitis B, diphtheria, pertussis, acellular pertussis, tetanus, and Hib, be eliminated from the market as soon as possible. Vaccine manufacturers must come up with a plan to replace existing vaccines with reduced mercury or mercury-free vaccines, and the FDA plans to expeditiously review revised vaccine product applications.

Thimerosal, which contains mercury, has been commonly used (since the 1930s) as a preservative in many vaccines, where it functions as an antimicrobial, especially in multidose containers. There is, however, a growing concern that the administration of multiple, sequential thimerosal-containing vaccines, especially to low-birthweight infants, could pose a risk. The agencies are quick to point out that there has been no firm clinical evidence of mercury toxicity in vaccine recipients, and children receiving these vaccines do not need to be tested for mercury exposure.

Until the availability of newer vaccines with a reduced level of mercury (or no mercury), the PHS and AAP are recommending that children continue to receive the usual childhood immunizations per the regular schedule, with the exception of hepatitis B vaccine. Since infants born to hepatitis B surface antigen-negative women do not require this vaccine, this vaccine can be deferred until 2-6 months of age when infants weigh more. The New Mexico State Department of Health has taken this recommendation a step farther and suggests that hepatitis B vaccine can be delayed until at least 1 year of age or until a mercury-free vaccine is available.

Both agencies are urging parents to continue to vaccinate their children, arguing that the known risk of inadequate immunization to potentially life-threatening childhood diseases outweighs any theoretical risk from vaccine administration. Nonetheless, it remains to be seen how this situation will affect the availability of certain vaccines over the next year. ■

Ehrlichia ewingii in Missouri: A New Human Pathogen

Source: Buller RS, et al. *N Engl J Med* 1999;341:148-155.

A causative agent of canine granulocytic ehrlichiosis, never previously identified in humans, has been identified in four patients presenting with fever in Missouri. The organisms were identified as an ehrlichia by "broad-range" PCR primers, but negative results were obtained when species-specific probes for the agents of human monocytic ehrlichiosis (due to *E. chafeensis*) and human granulocytic ehrlichiosis were used. Much to everyone's surprise, sequencing of 16 S-ribosomal RNA matched that of *E. ewingii*—a canine pathogen.

Interestingly, three of the four patients were receiving immunosuppressive therapy (including prednisone in two, and methotrexate and azathioprine in one each). All four responded to doxycycline.

Morulae were observed in neutrophils from two of the patients (originally misidentified as gram-negative coccobacilli), suggesting a diagnosis of human granulocytic ehrlichiosis, but serologies for this agent were negative. Western blots subsequently performed on specimens obtained from three of the patients during convalescence demonstrated cross-reactivity with one but not other major antigens of *E. chafeensis* and *E. canis*. In addition, serologies from one patient's dog were also consistent with recent infection due to *E. ewingii*.

Whether these cases represent accidental zoonotic transmission or a common vector remains unclear. Ehrlichiosis should be considered in any patient with an acute febrile illness and a culture-negative sepsislike syndrome who resides in an endemic area such as Minnesota, Wisconsin, Missouri, and the southeastern United States. As a former microbiology instructor of mine, Dr. George Sarosi, used to say, "No patient should die without the benefit of steroids." Perhaps this axiom should be updated to, "No patient should die without the benefit of steroids and doxycycline." ■

Rotavirus Vaccine on Hold

Source: *MMWR Morb Mortal Wkly Rep* 1999;48:577-581.

Based on concerns of a possible association between the administration of rotavirus vaccine (Rotashield) and an increased risk of intussusception in children, the American Academy of Pediatrics and the CDC are recommending that the use of this vaccine be suspended until the availability of additional safety data. Children scheduled to receive this vaccine, including those who have already begun the series, should not receive it until further notice. Any child who has recently received the vaccine and who develops gastrointestinal symptoms, including persistent vomiting, abdominal distention, severe abdominal pain, and black or bloody stools, should be urgently evaluated. Clinicians should be aware of this possible adverse event and are urged to report this or any other postvaccination event to the Vaccine Adverse Event Reporting System at http://www.nip.gov/nip/vaers.htm (1-800-822-7967). Data from an ongoing case-control study should be available by November 1999. ■