

Article originally published January 1999 in Well Beings—the now defunct newsletter produced by Sharon Kimmelman, founder of Vaccination Alternatives. This essay has been revised periodically. The date at the end indicates the last change or addition.

CHICKEN POX: *Why Do Children Die?*

By Gary Krasner

While chicken pox is rarely fatal, vaccination proponents in New York State want to mandate universal vaccination of school children against varicella. But rather than keeping them away from “infected” kids, Natural Hygienists suggest a better way to regain health and avoid death: Keep them away from allopathic physicians!

After learning this month of the legislative attempt to make the varicella vaccine mandatory in New York, I looked for a handle for an article. Since I didn't recall that chicken pox had ever been grouped in the category of medicine's infamous “Killer Diseases”, I thought I should find out how the Medical Boys justified making it compulsory for school children. It became apparent that the only medical justification for this vaccine had been the claimed mortalities. I went to the CDC's website and found something revealing in the May 15, 1998/Vol. 47/No. 18 issue of Morbidity and Mortality Weekly Report (MMWR, their official publication). It was entitled, “Varicella-Related Deaths Among Children: Texas and Iowa notified CDC of three fatal cases of varicella (chickenpox) that occurred in children during 1997” (reprinted in Appendix A below). A short introduction stated that in the U.S. there are approximately 100 deaths (about half of these in children) and 10,000 hospitalizations each year for complications from chicken pox from infection with the varicella virus.

After going over the report, I remembered why I stopped reading medical journals. In each of the three cases the young boys started out with fevers and/or other minor inflammatory conditions. Following each regimen of antibiotics, analgesics, or steroidal medications their condition grew progressively worse. The doctors responded to each new symptom with yet another drug, until the children died. Having an understanding of Natural Hygiene (note: it is briefly described by Harvey Diamond in his best seller, *Fit For Life*), I understood why the children got progressively worse from the drugging. But even equipped with a rudimentary understanding of the principles of N.H., one would realize that chicken pox is not a fatal disease, but rather a very common, benign inflammatory condition. And fatalities—as rare as they are—must actually result from inappropriate care, or the kinds of aggressive medical interventions described in the MMWR report.

With paraphrasing here and there, the next 9 paragraphs is taken from the section on chicken pox from the 1965 book, “*Food Is Your Best Medicine*” by Henry Bieler, M.D. He was a renowned clinician practicing in Pasadena, CA for over 50 years until his death in 1975. Dr. Bieler's skills were sought after by Hollywood celebrities and honored by his peers (a medical wing was named after him). His book is still available from Random House.

Chicken pox arises from the elimination of toxic fat or fatty acids through the hair fat glands. The chemical burn from the purging of waste products through the skin causes the characteristic blister of this disease. This occurs when the liver is congested and cannot perform its eliminative function and metabolic waste matter

(toxins) is then thrown into the bloodstream. These toxins in the blood must be discharged, so nature uses vicarious avenues of elimination, or “substitutes”. When these bile poisons (from the liver) in the blood come out through the skin, we get skin conditions manifested by rashes, boils, acne, etc. Or they come out through the mucous membranes (inside skin) manifesting as various catarrhs, like chicken pox. Thus, the skin is “substituting” for the liver, or a vicarious elimination is occurring through the skin.

FOOD AND DRUGS ARE CONTRAINDICATED

During the more acute and involved forms of toxemia, such as measles, chicken pox, fever, or flu, the liver is much too busy neutralizing toxic wastes to be bothered with digestion of food. Therefore, to facilitate the elimination of this waste, fasting on distilled water is essential in such cases. This accounts for the lack of digestive juices produced, and the loss of appetite that accompanies these illnesses.

After cells have been damaged by the toxic wastes, it is important for bacteria—acting as scavengers—to attack and devour the weakened, injured and dead cells. Otherwise, these dead cells would become accumulated toxic waste themselves. Therefore, antibiotics and other bactericides must not be administered. The so called “bad” bacterial strains die out on their own anyway, once their food (toxic waste) is used up. But until that point, they play an important role in the process that converts waste for eventual elimination.

Not only must germicides be withheld, but one must understand the other reason for fasting: Ingested food must either digest or decompose. Since digestion is diminished during these catarrhal illnesses, the latter holds sway, providing a growth medium for abnormal (read as “pathogenic”) bacterial strains.

The class of drugs that doctors use to treat catarrhal diseases are called antipyretics. Among antipyretics, aspirin tops the list of favorites. Aspirin is a phenol (carbolic acid) derivative, with all the chemical qualities of phenol, but without the deadly effect of carbolic acid. Aspirin, like phenol, deadens the nerve endings, thereby masking pain. But aspirin also diminishes a fever by partially blocking the thyroid and the adrenal glands (a bad thing). The phenol derivatives interfere with the proper function of the liver and damage liver cells. The use of aspirin, then, is an attempt to drive out one devil (disease toxins) by admitting another devil!

THE IMPORTANCE OF FEVER

Fever in a child is a frightening symptom to the mother. Just what is the function of fever? Is it a harmful process, something to suppress and worry about? Or is it the body’s attempt to burn up a poison, thereby helping to dispose of it more quickly?

In the diseases of childhood, fever begins in the liver. In a very strong, robust child, with properly functioning endocrine glands, the toxin is often completely consumed in the liver. The child does not feel sick or have pain; he just has a fever and if the liver area is carefully palpated, it can be noted that there is an elevation of temperature over that organ. In fact, if the temperature under the tongue is 105 degrees, the internal temperature of the liver may be as high as 110 degrees. But if the liver is unable to oxidize completely the poisons of disease so that some leak through into the blood stream, then, under the action of the endocrine glands, the poisons seek vicarious outlets via the mucous membranes. This may be through the upper respiratory tract, diagnosed by doctors as flu, sinusitis, pharyngitis, tonsillitis and possibly even pneumonia, which is a complicated kind of bronchitis. All through this process, the whole power of the liver is diverted into neutralizing the toxic wastes of disease, as evidenced by the fever.

The liver is much too busy to be bothered with the task of the digestion of food. Great strain can be taken off that organ if no food is given. Not only does fasting lower the temperature, relieve the distress and facilitate elimination, but it also lessens the strain on the liver and prevents serious complications, such as middle-ear

disease, mastoiditis and meningitis. Left alone, a fever will not exceed 106 degrees. And only about 4 percent of children experience fever-related convulsions, with no serious aftereffects.

A fast (on distilled water, or at least diluted fruit or vegetable juices) should be continued for twenty-four hours after the temperature has returned to normal. A good rule to remember is that the bowel can be cleared of toxins (by physic or enemas) in twenty-four hours; the blood in three days; the liver in five days, providing no food is eaten. Shingles (“adult chicken pox”), an eliminative crisis through the mucous membranes that occurs in adults, may require about a week-long fast to completely clear up. There should be little or no scars remaining, and absolutely no residual pain thereafter. [That was my experience with shingles 3 years ago.—G.K.]

It appears then, that fever, dreaded because misunderstood, is really nature’s attempt to help. It is discomfiting, but never does harm; never is attended with serious aftereffects and never should be suppressed with anti-inflammatory drugs or fed with food. I have seen many a case of flu pushed into a pneumonia because some anxious grandmother insisted upon something “to give the child strength”, such as chicken broth or a thin starchy gruel, both liquids, of course, but protein and starch—just what the liver cannot handle at this point.

THE TRUE CAUSE OF “INFECTIOUS” DISEASE

From Dr. Bieler’s words (above) we gain a little understanding of Natural Hygiene. So-called “infectious” diseases like chicken pox, measles, or whooping cough are actually inflammatory diseases. The symptoms during such illnesses should be viewed as eliminative crises. They may be very painful, but they’re a necessary self-limiting process in which an accumulation of retained metabolic waste (dead cells that become toxic), and the residues of undigested, unassimilated foods are being purged from the body through vicarious (abnormal; inappropriate) channels such as the skin or lungs. Thus, the familiar runny nose, cough, stiffness, fever, and numerous rashes, swellings, lesions, and eruptions through the skin are all manifestations of the same cause—which are *not* pathogenic microbes.

Microbes like bacteria, for example, act as scavengers to consume the toxic wastes and the dead cells following inflammation. Their formation and growth do not precede the diseased state in the host, but rather emerge in its wake; and not exogenically—from say, an “infected” person—but rather endogenically, from the genetic material contained in a cell’s nucleus after the cell’s death and decomposition. Fortunately, a wide range of bacterial strains, or their genetic “blueprints” (e.g., the various cellular and sub cellular—or “filterable”—stages that bacteria cycle through), inhabit our bodies all the time in titers low enough that their waste products do not affect us. Recently reported villains like salmonella, e. coli, or streptococcus are enteric and ever-present inside us. The viruses associated with measles, polio, influenza, and all the rest are also present—both in health and disease—and may have only an associative relationship with the diseases, but no proven causative roles. (Modern medicine still hasn’t determined the mechanism by which a virus causes poliomyelitis. When polio-like symptoms are present, and the virus is detected, the diagnosis is polio.) But when we become toxemic and our blood loses its alkalinity, the pathogenic strains begin to flourish in the bodily waste that accumulates—even well before any outward symptoms (inflammation and elimination) begin to appear. Their morphology (strain and function) is determined by the type of waste that is present for them to feed upon.

Symptoms are often triggered by a physiochemical or psychological “trauma”, such as exposure to cold or toxic chemicals, stress, lack of sleep, ingestion of spoiled meat, a sting or bite from an insect (hyper-reaction from the infection of foreign protein molecules), or an injected vaccine. Why these diseases occur predominantly in children was best described by Dr. Bieler: “The childhood years should be the healthiest of all. It is during those early years that the endocrine glands and the liver are in their best functional capacity, giving the healthy child his natural state of exuberance, inexhaustible energy, and faultless elimination”.

Elimination ends, whether treated with drugs, or untreated. When elimination ends and symptoms subside when treated with the former, doctors will proclaim that the drug had taken effect. But they are confusing symptoms with cause; believing that the disappearance of the former equates to the disappearance of the latter. But obviously a cause and an effect cannot be one in the same. When you stop the body from discharging toxic waste, you are not stopping the disease; you are merely stopping the effects.

In other words, neither allopathy, nor any other healing philosophy may claim responsibility for “curing” inflammatory or catarrhal diseases. Because the disease symptoms—the remedial actions initiated by our own bodies—themselves represent the “cure”.

But more importantly, when Allopathic physicians employ pain killers, fever suppressants, steroids and other drugs—which are sub-lethal doses of poisons—they have the effect of weakening the patient to the extent of checking elimination. This is a dangerous effect, because the waste products of these germs that have fed on the dead cells, together with the irritation from the toxins themselves may be absorbed into the blood, and irritating the already overworked liver—which is the detoxification center of the body. Antibiotics—which literally means “against life”—act chiefly by violently stimulating the adrenal glands. But if they are weak or depleted, the disease runs a chronic, often recurring course. In the aftermath of these germicides, there are also left fewer germs to convert waste, and no means to carry off and eliminate the dead cells. Not surprisingly, there are more deaths today from septicemia (blood poisoning caused by toxic waste from putrefactive bacteria) than there were before the use of antibiotics. (One of the boys from the MMWR report died from it.) Reactions from antibiotics include anaphylactic shock, aplastic anemia, and induced virulent infections. Deaths from penicillin still occurs today.

As Bieler wrote, fasting is the best therapy. Abnormal bacteria (strains that excrete toxic waste) evolve from, and proliferate on decaying matter. That’s what researchers use to culture pathogenic microbes. During illness, digestion is restricted to a degree where portions of ingested food will decompose in the gut, rather than digest. This provides a growth medium for harmful bacteria, and increases the toxic load on the body, which must summon energy to eliminate these endotoxins. Food concentrated with proteins, in particular, should be avoided until symptoms subside, as the byproducts of protein putrefaction are the most toxic.

CHICKEN POX DOESN'T KILL; DOCTORS KILL

It’s now plain to see why the children described in the afore-referenced MMWR had died. They were given numerous antibiotics, steroids, antipyretic and antipruritic medications and other fever suppressers, some administered directly into their bloodstreams. Probably they were given food to eat as well, even during the height of their inflammatory responses. The CDC admits that children don’t die from chicken pox per se, but rather “complications” from chicken pox. But what they don’t say is that these complications are all derived from acute blood toxemia established by the very treatments used by allopathic physicians. So strictly speaking, all children that die, do so from the allopathic medical treatments that are used to treat the symptoms that accompany chicken pox. There has never been a recorded death among the many thousands of children treated Hygienically, and without drugs.

What does the CDC list as the most common complication? Pneumonia and secondary bacterial infections (caused by the antibiotics). Other complications, according to the CDC, include encephalitis (inflamed brain tissue mostly from the antipyretics), hemorrhagic complications (such as intestinal bleeding, are the most common symptoms of aspirin—an anticoagulant, or “blood thinner”), hepatitis (congested and inflamed liver caused by the antipyretics), arthritis (decalcification of bone for the calcium needed to neutralize acidic blood, mostly caused by the aspirin), and Reye’s syndrome (most commonly associated with giving aspirin to children that have chicken pox or influenza).

Like aspirin and other anti-inflammatory drugs, acetaminophen (ie. Tylenol) will also burden the liver and kidneys and check the vital actions of the body to discharge waste from the blood. Acetaminophen poisoning is also common because it throws the chemistry of the liver off. In fact, it is the most common drug-induced cause of liver failure. It depletes hepatic glutathione, causing the toxic metabolite NAPQI to fail to conjugate, which leads to hepatic injury, and sometimes death.

Therefore, to say that “death is a complication of chicken pox”, is like saying, “bleeding is a complication of holding a knife in your hand”: each event is neither contingent nor a consequence of the preceding one. Their association is artificial; requiring specific intervening actions to take place. In cases of chicken pox, actions that are in accord and mandated by standard medical practice.

Fortunately, thousands of children each year get well *despite* being treated with drugs and fed food. But a handful do not.

To promote the vaccine, the CDC proclaims that, “varicella (chicken pox) is the leading cause of vaccine-preventable deaths in children in the United States.” But while the deaths are certainly preventable, they have nothing to do with the failure to vaccinate.

AUTHOR’S POSTSCRIPT:

The advice in this article is applicable to all inflammatory diseases. This article could have been titled, “Measles: Why Children Die”, or “Whooping Cough: Why Children Die”, etc. In each case, medications risk the life of the patient by checking the vital efforts of the body to eliminate waste through abnormal channels. Historically, Natural Hygiene had preceded allopathic medicine, and it represented a different paradigm of disease, particularly inflammatory (“infectious”) diseases. Hygienists would argue that allopathy’s perceived “success” in the prevention and cessation of physical symptoms is really achieved through “enervation”, or the weakening of the detoxification and eliminative capacity of our bodies through the use of sublethal dose of poisons (drugs). The (refuteable) claim that there’s a lower incidence of infectious diseases (just symptoms, mind you) among vaccinated children may simply prove that such children are more likely getting more drugs, vaccines and chemically laden food, which all contribute to enervation and symptom suppression. While many children may experience an eliminative crisis, it should not, by itself be fatal. To the contrary, such symptoms indicate that there’s a “cure” in progress, assuming that they’re left alone to run their natural course, unhindered and unmedicated.

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Two Books available from Foundation for Advancement in Cancer Therapies, Box 1242 Old Chesea Sta., New York, NY 10113. Make checks payable to FACT, Ltd. Add \$2 S&H. Add \$3 for first-class postage. Foreign orders: use postal money orders.

Food Is Your Best Medicine by Henry G. Bieler, M.D. Paperback, 1982 by Ballantine Books (236 pages). ____\$5.99. This book is also available from “www.randomhouse.com”.

Toxemia Explained by Dr. John Tilden. ©1976 by Keats Publ., New Caanan, CT. (130 pages). The theories of the successful clinician, John Tilden (1851-1940), who practiced conventional medicine for 18 years, then abandoned the use of all drugs to run a school and sanitarium in Denver. Describes toxemia as the basis of all diseases. ____\$5.50

DEBUNKING VIRUS DISEASES MAY BE FOUND AT:

www.soilandhealth.org (includes Natural Hygienic literature)

www.virusmyth.com

www3.bcity.com/harpub/

www.whale.to/vaccines

www.whale.to/disease_theory.html

www.garynull.com

www.sumeria.net

In 1997, 3 deaths reported by two states did not really occur from chicken pox, but rather from the unnecessary drugs they used to treat it. The preceding article refers to this CDC report, which is recorded here verbatim:

APPENDIX A:

Morbidity and Mortality Weekly Report

May 15, 1998 / Vol. 47/No. 18

Varicella-Related Deaths Among Children:

Texas and Iowa notified CDC of three fatal cases of varicella (chickenpox) that occurred in children during 1997:

CASE 1

On February 28, 1997, a previously healthy, unvaccinated 21-month-old boy developed a typical varicella rash. He had no reported exposure to varicella. On March 1, he was taken to a local emergency department (ED) with a high fever and was started on oral acetaminophen and diphenhydramine. On March 3, his primary-care physician prescribed oral acyclovir. On March 4, his mother noted a new petechial-like rash. The next morning, his primary-care physician noted lethargy, a purpuric rash, and poor perfusion. He was transferred to a local ED. Fluid resuscitation and intravenous ceftriaxone were initiated, but the child continued to deteriorate rapidly, requiring intubation, mechanical ventilation, and inotropic support with dopamine. Blood cultures were negative for bacterial pathogens. Laboratory tests indicated disseminated intravascular coagulation and severe dehydration. Approximately 1.5 hours after arrival at the ED, he was transported to a tertiary-care center. Within 10 minutes of arrival, he suffered cardiac arrest and died. The death was attributed to varicella with hemorrhagic complications.

CASE 2

On December 21, 1997, a 5-year-old unvaccinated boy with a history of asthma was taken to a local ED with a fever of 104.5 F (40.3 C) and a typical varicella rash in multiple stages of healing. The child was treated with antipyretic and antipruritic medications and discharged.

That evening, the boy developed mild dyspnea and was treated at home for a presumed asthma attack with metered-dose inhalers and one dose of oral prednisone. He returned to the ED on December 22 with shortness of breath and a 4-hour history of abdominal and leg pain. On presentation to the ED, one of the patient's siblings had active varicella and another had recently recovered from varicella. Physical examination revealed numerous chickenpox lesions, one of which appeared infected. He was tachypneic, and his extremities were mottled consistent with peripheral septic emboli. Chest and abdominal radiographs revealed a right pleural effusion, pneumonia, and mild ileus. Thoracostomy produced pleural fluid containing gram-positive cocci, confirmed 8 hours later to be group A Streptococcus (GAS). A peripheral blood sample revealed gram-positive cocci. He was admitted to the hospital and treated with intravenous ceftriaxone, nafcillin, and acyclovir.

After admission, his breathing became labored and his extremities increasingly mottled. He rapidly developed hypotension, obtundation, and bradycardia. Despite efforts at cardiopulmonary resuscitation, the child died 5 hours after arriving at the ED. A post-mortem examination attributed the death to GAS septicemia, pneumonia, and pleural effusion, complicating varicella infection.

CASE 3

On December 14, 1996, a previously healthy, unvaccinated 23-month-old boy developed fever and a typical varicella rash. Approximately 1-2 weeks earlier, his unvaccinated 4-year-old sibling had contracted varicella. He was taken to his physician on December 17 because of persistent fever and cellulitis of the left foot, and he was hospitalized on December 19 for failure to improve on an unspecified outpatient antibiotic regimen. Because his condition deteriorated despite intravenous methicillin and ceftriaxone, he was transferred to a regional hospital on December 21. Sepsis, possible viral meningoencephalitis, and mild pleural effusion were diagnosed. A cerebrospinal fluid examination revealed lymphocytic pleocytosis, and blood and urine cultures grew penicillin-resistant *Staphylococcus aureus*. Antibiotics were changed to nafcillin and gentamycin, and intravenous acyclovir was added on December 23. On December 24, the child developed an aortic insufficiency murmur, and an echocardiogram revealed a 9x9 mm vegetation on the aortic valve, consistent with bacterial endocarditis. Serial echocardiograms displayed growth of the vegetation and development of a pericardial effusion. He was transferred to a cardiac surgery center on December 26. While awaiting surgery, he developed refractive heart failure secondary to staphylococcal endocarditis. He became incoherent, probably secondary to a major embolic neurologic event, and died on January 8, 1997.

PUBLISHER'S POSTSCRIPT

We received positive responses from many who read, "Chicken Pox: Why Do Children Die?", from our 11/98-1/99 double issue. The following letter from Benjamin Estrada, M.D. published in "Infections in Medicine®" [*Infect Med* 16(5):307, 1999. © 1999 SCP Communications, Inc.] apparently supports Gary Krasner's assertion that the complications that children die from are not caused by chicken pox per se, but rather from the the drugs that doctors use to "treat" chicken pox. —*Sharon Kimmelman, Publisher, WB.*

Pediatric Bulletin Varicella and GAS: Do NSAIDs Fuel the Fire?

Author: Benjamin Estrada, MD, University of South Alabama, Mobile, Ala.

Published in: *Infect Med* 16(5):307, 1999. © 1999 SCP Communications, Inc.]

During the past decade, there has been an increase in the frequency of severe Group A beta-hemolytic streptococcal (GAS) infections in children. Factors associated with this development are an increase in the prevalence of exotoxin-producing serotypes and low herd immunity. The increase is due in part to the low rates of infection with these strains in the past.

It has been noted in some series that severe invasive GAS infections such as necrotizing fasciitis (NF) and streptococcal toxic shock syndrome (STSS) are associated with preexisting varicella infections in up to 47% of patients (Peterson CL et al: *Pediatr Infect Dis J* 15:151-156, 1996). Another possible association, this one between the use of nonsteroidal anti-inflammatory drugs (NSAIDs) and severe GAS infection in children with varicella, has also been reported by several investigators. NSAIDs have been used to ameliorate the signs and symptoms of varicella, but the question of whether their use increases disease progression remains.

The association between fulminant NF and the use of NSAIDs was reported by Rimailho and collaborators more than a decade ago (Rimailho et al: *J Infect Dis* 155:143-146, 1987). These investigators described fulminant disease in five patients treated with NSAIDs, which included aspirin, diclofenac, piroxicam, and niflumonic acid. Several studies have demonstrated the development of lymphopenia and decreased lymphocyte function in the presence of aspirin and other NSAIDs. It has also been shown that abnormal neutrophil chemotaxis, chemiluminescence, and lymphocyte transformation of PHA occurred when leukocytes from a patient with NF were exposed to NSAIDs. This information suggests that NSAIDs may decrease immune function and favor a widespread infection in patients infected with invasive strains of GAS (Smith RJ: *South Med J* 84:785-787, 1991).

The association between the use of ibuprofen and the development of severe GAS infection in children with varicella was first reported by Brogan and colleagues (Brogan et al: *Pediatr Infect Dis J* 14:588-594, 1995) in a series in which five children developed GAS NF while receiving treatment with this NSAID. The investigators concluded that it may be prudent to limit the use of this drug for local complications of varicella, since it may impair granulocyte function and at the same time mask the signs of disease progression with GAS.

Until recently, most of the evidence suggesting a potential association between the use of ibuprofen in patients with varicella and the development of GAS invasive disease was based on isolated case reports or data obtained from small case series. A recent study aimed at the evaluation of risk factors associated with the development of invasive GAS infection in patients with varicella found that the development of invasive disease with this bacteria was 8.3 times more likely in those patients in whom ibuprofen had been used during the first 5 days after the onset of varicella (Peterson CL et al: *Pediatr Infect Dis J* 15:151-156, 1996).

More recently, a case-controlled study was performed to determine whether ibuprofen use was associated with the development of NF in patients with varicella. This study included 19 children with varicella and NF and 29 controls also diagnosed with varicella and a serious soft-tissue infection other than NF. Ibuprofen use before hospitalization was more likely in cases than in controls (42% vs 15%). Patients with NF complicated by renal insufficiency or STSS were also more likely to have used ibuprofen than those with uncomplicated NF. Although this study does not establish a direct causal relationship between ibuprofen use and the development of GAS NF in patients with varicella, the findings imply that an association may exist. The authors suggest that this association could either be due to a more severe GAS infection promoted by the immunoinhibitory effect of ibuprofen, or “masking” of the signs and symptoms of disease progression by the action of the same drug. Another possibility is that ibuprofen use could be only an indicator of more severe disease that required more aggressive anti-inflammatory management (Zerr DM et al: *Pediatrics* 103:783-790, 1999).

These studies suggest that there is an association between the use of ibuprofen (and possibly other NSAIDs) in children with varicella and the development of severe invasive GAS infection. Until a definite causal relationship can be established or ruled out by future studies, practitioners should consider the potential risks of using these medications in children with varicella. Providing comfort for symptom relief through the administration of these drugs must be weighed against the potential for development of severe GAS disease.

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ADDITIONAL CASES OF DEATHS RESULTING FROM TREATMENT FOR CHICKEN POX

Varicella-related deaths--Florida, 1998. MMWR Morb Mortal Wkly Rep. 1999 May 14;48(18):379-81.PMID: 10369579; UI: 99296270
<http://www.cdc.gov/mmwr/preview/mmwrhtml/mm4818a3.htm>

Case 1. On February 19, a healthy, unvaccinated 6-year-old boy developed a varicella rash, abdominal pain, malaise, and loss of appetite following exposure to a classmate with varicella. The child had asthma and intermittently had been on inhaled steroid therapy but had not received steroids within the previous month. On February 22, he was hospitalized with hemorrhagic skin lesions, tachycardia, tachypnea, and a platelet count of 89,000 (normal range: 150,000-350,000). Several hours after admission he developed pulmonary edema and respiratory insufficiency and required mechanical ventilation. He died on February 23. Tissue samples of multiple organs had a positive polymerase chain reaction for varicella zoster virus (VZV).

Case 2. On March 27, a healthy, unvaccinated 58-year-old woman developed a varicella rash. She was born in Cuba and had moved to the United States in 1995. She did not have a history of or known exposure to varicella. On April 3, she was hospitalized with a 5-day history of increasing shortness of breath and productive cough and was diagnosed with varicella pneumonitis. She was treated with intravenous acyclovir and ceftriaxone, but developed adult respiratory distress syndrome (ARDS), disseminated intravascular coagulopathy, renal failure, and coma. She died on April 20.

Case 3. On April 27, a healthy, unvaccinated 29-year-old man developed a varicella rash. In early April, his children had contracted varicella. On April 29, he sought care at a local emergency department for chest pain and respiratory distress. Chest radiographs showed bilateral pulmonary interstitial infiltrates. On April 30, he began coughing up blood, was intubated because of increasing respiratory insufficiency, and was treated with intravenous acyclovir and antibiotics. He developed sepsis, ARDS, and multiorgan failure, and died May 12.

Case 4. On May 5, a 21-year-old unvaccinated female employee at a family child care center developed a varicella rash after exposure to a child with varicella. The employee had a history of asthma and was treated with 5 mg prednisolone per day. She was hospitalized on May 7 with varicella pneumonitis and received intravenous acyclovir on May 8, but she died the same day.

Case 5. On July 11, an 8-year-old unvaccinated boy developed a maculopapular rash diagnosed clinically as varicella and confirmed by direct fluorescent antibody test on July 23. He had acute lymphocytic leukemia (ALL) and had been on immunosuppressive therapy since receiving a bone marrow transplant on May 15. He had not had varicella and had no known varicella exposure. He was treated with varicella zoster immunoglobulin on July 16 and acyclovir on July 23. He died on July 25 after recurrence of leukemia with a graft-versus-host reaction complicated by disseminated varicella, cellulitis, ileus, and hypertension.

Case 6. On October 3, an unvaccinated 45-year-old man with diabetes mellitus, asthma, and cirrhosis of the liver developed a varicella rash. He was born in Cuba and had resided in the United States for 35 years. He had no history of varicella and no known exposure. He was not receiving steroids or immunosuppressive drugs. He was admitted to the hospital with varicella on October 5 and on October 6, treatment was initiated with oral acyclovir. He died on October 8; pathologic evidence from the postmortem examination revealed VZV in all major organs.