Toxic Shock-Like Syndrome

These severe infections produce what is called a toxic shock-like syndrome that includes necrotic, soft-tissue infections, shock, kidney dysfunction, and acute respiratory distress. Mortality may be as high as 30%.

People who have antibodies against the virulence factors, such as toxins produced by the bacteria, do not develop the disorder.

"Certain host cells, such as the monocytes and macrophages, serve as the battleground between the invading bacteria and the host. If the secreted strep toxins reach these cells, they overreact," Stevens said. If enough cells are affected, the patient dies.

"This explains why in severe infections or in cases where the patient presents late, such as puppeteer Jim Henson, there is a grave prognosis in spite of appropriate antibiotics and supportive care," he said, noting that his research group is investigating novel ways to combat such cases.

In 1989, Stevens reported a study of 20 patients from the Rocky Mountain region who had group A streptococcal infection that was remarkable for the severity of local tissue destruction and life-threatening systemic toxicity. Strains of group A β-hemolytic streptococci isolated from eight of the patients produced pyrogenic exotoxin A, which has rarely been found in group A strep in recent years.

Since that publication, nearly 30 reports have been published or presented describing similar infections in young patients in Western Europe, Australia, Scandinavia, and the United States, he noted.

M Protein

The M protein is responsible for much of the damage caused by streptococcal infection. The M protein is known as a superantigen because of its ability to stimulate immune cells and provoke a powerful immune response.

The protein is anchored within the bacterial wall by a group of six amino acids, a site for potential therapeutic intervention.

Vincent A. Fischetti, MD, of the Rockefeller University in New York, and his colleagues have isolated the group of six amino acids, which they call the Achilles' Heel in the M protein on the surface of group A streptococci. In addition to strep, this same group also is found in a variety of other gram-positive bacteria.

"This group provides a potential new target for antibiotic intervention," Fischetti said. "Just as penicillin and related antibodies interfere with the assembly of the bacterial cell wall, an alternative antibiotic strategy may now be developed to block the attachment of cell surface proteins in gram-positive bacteria. And if you block the way these proteins are attached, you have a good chance of eliminating the organism's ability to cause disease."

Malak Kotb, MD, reported that she and her colleagues at the University of Tennessee, Memphis, Tennessee, have discovered a novel mechanism by which M protein stimulates some human lymphocytes.

Continuing work begun by the late Edwin H. Beachey, she and her colleagues have found that the M protein stimulates immune response cells that are normally active. Although many of the stimulated cells die, others appear to migrate to body tissues such as the heart or kidneys, where they damage those organs.

"In the past, such damage had been attributed to antibodies produced by the body to tight off strep bacteria, but it now appears that the damage results from the M protein's stimulation of immune response cells," she said.

Both studies were reported at the annual meeting of the American Society for Microbiology in Dallas, Texas.

From *Infectious Disease News.* July 1991;4:20.

CMV-IG Available for Recipients of CMV-Seropositive Kidneys

Connaught Laboratories, Inc. (Swiftwater, Pennsylvania) is marketing a prophylactic intravenous cytomegalovirus (CMV) immune globulin (CMV-IG) for seronegative recipients of seropositive kidneys. In a randomized trial,[1] the incidence of virologically confirmed CMV-associated syndromes was reduced from 60% in controls (n = 35) to 21% in recipients of CMV-IG (n = 24) ($p < .01$); marked leukopenia was reduced from 37% in controls to 4% in recipients (n = 24 ($p < 01$); and fungal or parasitic superinfections were not seen in CMV-IG recipients but were seen in 20% of controls ($p = .05$).

CMV, a herpesvirus spread by close contact with infected secretions or the introduction of infected blood or organs into a host, can cause severe problems for kidney transplant patients. CMV can cause retinitis (which can lead to blindness), pneumonia, hepatitis, and may contribute to graft rejection.

REFERENCE

1. Snydman DR, Werner BG, Heinze-Lacy BH, et al. Use of cytomegalovirus immune globulin to prevent cytomegalovirus disease in renal transplant recipients. *N Engl J Med.* 1987;317:1049-1054.