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Commentary

Infectious Outbreaks in the Immunocompromised Host

Candella Abril*

Department of Infectious Disease, National University of Rosario, Rosario, Argentina

ABOUT THE STUDY

The innate immune system provides largely nonspecific protection against the initiation of infections. Its components include epithelial barriers, phagocytes, complement and natural killer cells. The adaptive immune system mediates targeted defense against repeated infections. The two arms of this system compromise humoral immunity, directed against extracellular organisms and cell-mediated immunity, directed against intracellular organisms.

Immunocompromised host

The term "immunocompromised host" encompasses a group of patients with diverse and often overlapping conditions which impair various host defense mechanisms and decrease the patients' ability to respond to infectious diseases. The normal host defenses include the cells of the immune system, immunoglobulins, complement and various biochemical and physical barriers. Compromise of one or more of these components predisposes the host to a range of infections, depending on the nature of the defect or defects. Focal defects may result in specific susceptibilities, for example splenic dysfunction predisposes the patient from infections with encapsulated bacteria. In other cases, broad impairment of many components of host defense, such as occurs following bone marrow transplantation, results in susceptibility to infection by a wide range of organisms. The acquired immune deficiency syndrome gives rise to progressive immunocompromise following HIV infection, which predisposes patients to a range of opportunistic infections including cryptococosis and toxoplasmosis.

Anatomical barrier defects

Physical barriers to infection include the epithelial of the skin, gastrointestinal tract and respiratory tract. These epithelia are associated with substances, secretions and specialized cells with antimicrobial properties. In addition, the barriers are colonized by the commensal microorganisms in symbiosis with the host. The physical barriers to infection may be disrupted by trauma, surgery and the insertion of catheters or other foreign bodies. The integrity of the barriers may be impaired by treatments including chemotherapy and antibiotic therapy which removes the commensal flora permitting colonization with potentially more virulent organisms. Anatomical barrier defects predispose patients to infections with skin colonizing organisms such as staphylococci and gastrointestinal tract commensals such as streptococci and Pseudomonas species.

Neutropenia

Neutropenia is defined as an absolute neutrophil count of less than 500 cells/mm^3 or less than 1000 cells/mm^3 but not expected to decrease to less than 500 cells/mm^3 within 48 hours. Neutropenia may arise as a consequence of hematological disorders including acute leukemia and aplastic anemia. The use of cytotoxic chemotherapy for the treatment of hematological and solid organ malignancies gives rise to severe and prolonged periods of neutropenia.

As neutrophils provide the primary defense against bacterial and some fungal infections, neutropenia predisposes patients to these infections. There is a linear relationship between both the severity and duration of neutropenia and incidence of infections. Most infections are caused by bacteria or fungi from the patient's endogenous flora, the majority arising from the flora of the gastrointestinal tract. Until the early 1980s, most infections in neutropenic chemotherapy patients were caused by Gram negative bacteria, but since this time Gram positive bacterial infections have begun to predominate over Gram negative infections. Reasons for this shift include the increased use of intravascular devices and the use of prophylactic antibiotics with extensive Gram negative cover such as ciprofloxacin. Early course of neutropenia, most infections are bacterial in origin with the emergence of fungal infections usually occurring in the setting of prolonged and profound neutropenia.

Humoral immune system dysfunction

The humoral immune response is primarily directed against extracellular bacteria. Components of the humoral immune system including complement and antibody facilitate the

Correspondence to: Candella Abril, Department of Infectious Disease, National University of Rosario, Rosario, Argentina, E-mail: brill@ca2ndella.ar

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internalization and killing of extracellular pathogens by the cells of the innate immunity system. Specific antigens give rise to the activation of subsets of B cells. The B cells present these antigens to CD4 T cells, leading to complement-mediated lysis or phagocytosis. B cells are also capable of differentiating into antibody producing plasma cells. Secretory immunoglobulin impairs bacterial motility and adhesion to epithelial surfaces. Immunoglobulin production is decreased in chronic leukemia and multiple myeloma resulting in increased susceptibility to infection by extracellular bacteria, particularly encapsulated organisms such as *Streptococcus pneumoniae* and *Haemophilus influenzae*.

Splenic dysfunction

An absent or poorly functioning spleen leads to an increased incidence of several bacterial infections, giving rise to postplenectomy sepsis. Functional hyposplenism may occur in a range of autoimmune hematological, neoplastic, infiltrative and gastrointestinal disorders and as a consequence of advanced age, malnutrition and alcohol excess. The immunological functions of the spleen leads to an include antibody production and the clearance of encapsulated organisms from the bloodstream. Postsplenectomy sepsis is most often caused by encapsulated organisms such as *S. pneumoniae* and *H. influenzae*. Postsplenectomy typically presents with a short, prodomal febrile illness followed by rapid progression to sepsis shock with a high mortality rate. The prompt administration of emperical, broad spectrum antibiotics with activity against encapsulated organisms may be lifesaving. Pediatric patients are often given prophylactic oral penicillin V for several years following splenectomy. Such treatment is also used for some adults following splenectomy. Asplenic and hyposplenic patients should be offered immunization with the pneumococcal polysaccharide vaccine, the *H. influenzae* Type B vaccine, the quadrivalent meningococcal vaccine and yearly trivalent inactivated influenza vaccine immunizations.