Multiple Myeloma: An Overview

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Multiple myeloma (MM) is a malignant hematologic disorder involving plasma cells. In MM, immunoglobulin is overproduced, and patients can develop skeletal, hematologic, renal, and/or neurologic complications. The exact etiology of MM is unknown. The treatment for MM includes chemotherapy, antiangiogenic medications, and, most recently, a proteasome inhibitor. Nursing care for patients with MM requires close monitoring for infections and anemia, pain control, and education about the disease and treatment options. Further understanding of the pathophysiology of MM may lead to newer treatment options for the disease.

Key Words: multiple myeloma, thalidomide, antineoplastic agents

Multiple myeloma (MM) is a plasma cell neoplasm accounting for 1% of all malignancies and 14% of malignant hematologic disorders (Jemal et al., 2004). It is characterized as a B cell malignancy with an increased production of one of the immunoglobulins and can present with skeletal, hematologic, renal, and neurologic complications. Compared to other hematologic malignancies, MM is less common, but recent developments of new therapies have heightened awareness and education. This article addresses etiology, pathophysiology, signs and symptoms, treatment options, and nursing implications.

Etiology, Incidence, and Risk Factors

About 15,270 new cases of MM are diagnosed each year, and MM causes approximately 11,070 deaths per year (Jemal et al., 2004). The annual incidence rate is about 4 per 100,000 people, with the incidence for African Americans almost twice that of Caucasians (Zaidi & Vesole, 2001). The median age at diagnosis is 65 years, and fewer than 3% of patients are younger than 40 years (Blade & Kyle, 1998).

The exact etiology of MM remains unknown. Although several risk factors are thought to be associated with the disease, no evidence exists to suggest a hereditary basis (George & Sadovsky, 1999). Exposure to ionizing radiation and pesticides (such as dioxin) has been identified as a risk factor for MM (Durie, 2001). MM is more common in farmers, wood and leather workers, and those exposed to petroleum products (Sheridan, 1996). Viruses such as human herpes 8 and simian virus 40 have been described in the pathogenesis of MM (Rettig et al., 1997). In addition, exposure to Agent Orange and HIV have been associated with MM (Zaidi & Vesole, 2001). Chromosomal translocations typically are seen in MM cell lines (Kaufmann, Urbauer, Ackermann, Huber, & Drach, 2001). Prevention measures may be forthcoming when etiologies of MM are more fully understood.

Pathophysiology

Knowledge of MM requires an understanding of normal hematopoesis. All immune cells begin in the pluripotent stem cell, which has the ability to replicate or differentiate into either lymphoid or myeloid lineages. The myeloid lineage further differentiates into platelets, neutrophils, eosinophils, and basophils; the lymphoid stem cell divides into either T or B lymphocytes. B lymphocytes mature into immunoglobulin-producing plasma cells, which are responsible for humoral immunity. Each immunoglobulin is a protein consisting of four polypeptide chains, two light chains and two heavy chains. The light chains are kappa and lambda, and the heavy chains define the five classes of immunoglobulins: IgG, IgA, IgM, IgE, and IgD. Each immunoglobulin has a particular role and function in the immune response (see Table 1). In MM, an abnormal overproduction of one of these immunoglobulins occurs, with IgG being the most common (Harousseau, 2002) (see Table 1). This overproducing protein is referred to as the M protein, meaning the monoclonal protein or the myeloma protein.

The establishment of MM cells in bone marrow involves a complex interaction with the bone microenvironment. MM cells establish themselves by binding to bone marrow stromal cells, giving them a growth and survival advantage. This advantage comes from resistance to drug-induced apoptosis and an interaction leading to increased cytokine and interleukin-6 (IL-6) secretion. IL-6 has proven to be a significant MM growth and survival factor (Anderson, 2001, 2002). Current research efforts and novel therapies in MM are targeting this interaction between the bone microenvironment and MM cells. The advancement of novel therapies will rely on the pathophysiology and further understanding of the etiology of the disease.

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