Monoclonal gammopathy of undetermined significance—Making It Understandable to Patients

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Monoclonal gammopathy of undetermined significance (MGUS) was first described by Jan Waldenström in 1960 after abnormal narrow hypergammaglobulinemia bands were noted on serum protein electrophoresis in the blood of healthy individuals (Kyle, Buadi, & Rajkumar, 2011). Robert Kyle used the term “monoclonal gammopathy of undetermined significance” in 1978 after detecting a correlation between MGUS and the development of other disorders such as multiple myeloma, Waldenström’s macroglobulinemia, amyloidosis, and chronic lymphocytic leukemia (Korde, Kristinsson, & Landgren, 2011). Therefore, MGUS is defined as the presence of an abnormal protein (monoclonal protein or M protein) in the blood or urine in the absence of end-organ damage. In 2010, the International Myeloma Working Group defined MGUS as a serum monoclonal protein under 3 g/dl with fewer than 10% clonal bone marrow plasma cells and an absence of end-organ damage, which can be attributed to the plasma cell proliferative disorder, examples include hypercalcemia, renal insufficiency, anemia, and bone lesions (Kyle et al., 2010).

Prevalence and Risk

A systematic review of 14 studies estimated that the prevalence of MGUS was about 3% in people older than 50 (Wadhera & Rajkumar, 2010). The prevalence increases with age and is estimated to be 7% in Caucasians older than 80. Additional risks include race (6% in Caucasians compared to 8% in African Americans) (Greenberg, Vachon, & Rajkumar, 2012), gender (4% of Caucasian men older than 50 years compared to 3% of Caucasian women), family history (Greenberg, Rajkumar, & Vachon, 2012), and immunosuppression. For immunosuppressed patients with HIV, a substantial risk exists ranging from 9%–45% (Wadhera & Rajkumar, 2010). Infection also has been associated with the development of MGUS. Sixty-eight percent of patients with MGUS also have had Helicobacter pylori infections, and the eradication of the infection led to resolution of monoclonal gammopathy in 11 of 39 patients (Kyle et al., 2011). Environmental exposure to toxins such as asbestos, pesticides, radiation, petroleum, aromatic hydrocarbons, fertilizers, and paints poses additional risk. However,