FEATURE ARTICLE

Recognizing Hyperviscosity Syndrome in Patients With Waldenstrom Macroglobulinemia

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Hyperviscosity syndrome can develop in patients with plasma cell dyscrasias, particularly Waldenstrom macroglobulinemia (WM). Occurring in 10%–30% of patients with hyperviscosity syndrome, WM is an uncommon B-cell proliferative disorder characterized by bone marrow infiltration and production of monoclonal immunoglobulin M. The elevated blood viscosity in WM is the result of increased circulating serum immunoglobulin M. Because hyperviscosity syndrome can be lethal, it must be recognized and managed early. Hyperviscosity syndrome has a triad presentation: vision changes, neurologic abnormalities, and bleeding. Treatment includes hydration with diuresis, plasmapheresis, and control of the underlying disease. The current treatment for WM is chemotherapy (i.e., alkylating agents and nucleoside analogs) and the monoclonal antibody rituximab. Although hyperviscosity syndrome is not one of the most common conditions, when it does occur, oncology nurses play a critical role in patients' assessment and care.

Ms. C is an 80-year-old woman who was diagnosed with Waldenstrom macroglobulinemia (WM) after being hospitalized twice for acute confusion. One of the episodes was severe enough to require admission to an intensive care unit. Ms. C has a history of coronary artery disease and status postendarterectomy two years ago, hypertension, and chronic renal insufficiency. Her confusion was believed to be related to cardiac problems or "mini strokes," but her cardiac function showed normal ejection fraction. She underwent computed tomography and magnetic resonance imaging scans of the brain, which were negative for acute process.

On Ms. C's second admission, her family reported that she complained of increasing fatigue and confusion. During the workup, she was anemic and received a blood transfusion. Because of the anemia and fatigue, malignancy was a concern; therefore, an additional workup was initiated. Ms. C's chemistries showed elevated total protein and mild elevation of calcium. Immunoglobulin (Ig) M was elevated with depressed IgG and IgA. The workup included a serum protein electrophoresis that showed an IgM kappa paraprotein peak of 3.5 g/dl and urine immunofixation that had 14 mg of kappa Bence-Jones proteinuria.

Ms. C was referred to an oncologist who obtained a bone marrow biopsy that revealed B-cell lymphoplasmacytic population with monotypic kappa chains expressing IgM that was consistent with the diagnosis of WM. Ms. C sought a second opinion for confirmation of her diagnosis and treatment recommendations. At the time of her visit, she denied any fever, chills, or night sweats. She reported weight loss of approximately 15 pounds in the previous six months and denied having any neurologic problems, bleeding, or visual disturbance. She had elevated blood pressure (190/110 mmHg), and a funduscopic examination revealed mild retinal vein tortuosity and retinal hemorrhages in the right eye.

At a Glance

- The clinical presentation of hyperviscosity syndrome is not limited to vision and neurologic abnormalities and mucosal bleeding; cardiopulmonary symptoms, acute renal failure, and stroke also have been reported.
- Hyperviscosity symptoms appear when the viscosity level reaches 4–5 cp.
- Nursing implications for patients with hyperviscosity syndrome should include education, symptom management, and supportive care.

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