Oral chronic graft-versus-host disease (GVHD) is a serious complication experienced by patients following allogeneic hematopoietic stem cell transplantation (HSCT) that contributes to morbidity and mortality (Imanguli, Pavletic, Guadagnini, Brahim, & Atkinson, 2006). GVHD develops when grafted immunocompetent T cells (transplanted stem cells) recognize and reject foreign antigens on the host (recipient) cells, leading to a T cell-mediated inflammatory reaction (Morton & Fontaine, 2011). Acute GVHD usually manifests within 100 days following HSCT, and chronic GVHD (cGVHD) generally develops more than 100 days following HSCT. cGVHD occurs in 30%–60% of patients surviving more than 100 days postallogeneic HSCT, and may present as late as one year after HSCT (Lee & Flowers, 2008; Morton & Fontaine, 2011). It typically occurs in patients who have had acute GVHD. Reduced relapse rates have been seen in allografted patients who developed GVHD when compared with patients who did not develop the disease. The graft-versus-tumor effect reduces relapse rates because host antigen-presenting cells during the first months after HSCT cause efficient donor T cell immunization against host hematopoietic cells (Baron et al., 2005).

The most frequent sites involved in cGVHD at time of diagnosis are the skin, mouth, liver, and eyes (Lee & Flowers, 2008). Standard treatment of cGVHD consists of systemic immunosuppressive therapy, usually with steroids, for periods of two to seven years. That therapy, in addition to the chronic immunodeficiency and organ damage caused by the cGVHD, contributes to increased morbidity and mortality (Lee & Flowers, 2008). Although optimal treatment is not yet available for cGVHD manifesting in the oral cavity, patients with oral cGVHD need to receive the best available therapy to decrease disease severity and related symptoms, as well as to minimize the associated negative effects on their health-related quality of life (HRQOL) and nutritional status (Fall-Dickson et al., 2010).

Clinical Presentation

Oral involvement often is the first manifestation of the disease (Imanguli et al., 2006). Almost 80% of patients with cGVHD have oral clinical signs, including atrophy, edema, erythema, lichenoid changes, ulcerations, and late fibrosis, which often lead to poor jaw range of motion (Imanguli et al., 2006). Lichenoid changes are similar in clinical presentation to oral lichen planus and have a