Herpes Simplex Virus–Related Oral Mucositis in Patients With Lymphoma

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A 58-year-old man named J.S. was diagnosed with non-Hodgkin lymphoma and underwent treatment with standard rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone (R-CHOP) chemotherapy. He presented to his local outpatient clinic for evaluation and laboratory tests on day 10 after cycle 3. During this visit, J.S. reported great difficulty opening his mouth with significant gingival and lingual pain when eating and drinking in spite of prophylactic oral care. Laboratory test results revealed a white blood cell count of 0.9 k/ul, hemoglobin level of 8.9 g/dl, platelets of 100 k/ul, serum creatinine level of 1 mg/dl, and blood urea nitrogen level of 29 mg/dl.

Examination revealed a man in good physical shape with normal vital signs and oxygen saturation level on room air. Although his oral mucosa was pink, his tongue and buccal mucosa had bilateral ulcerations with xerostomia (dry mouth). Upper and lower gingivae were intact but sensitive to touch. The remainder of his physical examination was normal. He was provided with education on continuing oral care, antivirals, analgesics, and neutropenic precautions. He did receive growth factor support 24 hours after completion of therapy. Diagnosis based on clinical presentation was herpes simplex virus (HSV)-related oral mucositis.

Oral Mucositis

Studies have investigated the incidence and management of oral mucositis as well as its significant consequences, which may occur as a complication of mucositis. However, limited information exists on the incidence of oral mucositis resulting from a viral etiology such as HSV (Chen et al., 2011). No clear evidence supports routine cultures or empiric treatment with antivirals for presumed HSV-related oral mucositis. Healthcare professionals must identify, assess, and manage oral mucositis and should be aware that HSV infection is a potential cause or complication of oral mucositis.

Oral mucositis is an inflammatory process in the oral mucosa often caused by chemotherapy agents or radiotherapy. Oral mucositis is characterized by clinical signs and symptoms observed in the oral cavity. In the general oncology population, oral mucositis ranges from mild sensations in the oral cavity to a myriad of confluent ulcerative lesions. Mucositis can affect the continuum of the oral and gastrointestinal mucosa, from the mouth to the anus (Eilers & Million, 2007). For purposes of this case study, the authors will focus on the oral cavity.

Incidence

Oral mucositis occurs frequently in the general oncology population. Sonis (2004) reported that the incidence of oral mucositis can be as high as 100%, depending on the patient characteristics and treatment regimens. Vera-Llonch, Oster, Ford, Lu, and Sonis (2007) reported that 51% of patients receiving standard chemotherapy for lymphoma develop oral and/or gastrointestinal mucositis. The degree, extent, and site of oral mucositis that develops in any particular patient may depend on factors such as age, underlying systemic disease, and race, as well as tissue-specific factors (e.g., epithelial types, local microbial environment, function). In addition, conflicting evidence exists for the influence of gender on risk for mucositis, with some studies reporting increased risk for mucositis in females and others finding no gender effect (Lalla, Sonis, & Peterson, 2008). Smoking, obesity, cachexia, history of prior chemotherapy, and poor oral hygiene and dentition also place patients at increased risk of oral mucositis (Eilers & Million, 2007). Genetics also may play a role in mucositis development (Cawley & Benson, 2005).

Pathophysiology

Oral mucositis was previously considered a simple process because of the non-specific damage to epithelial cells by cytotoxic therapies and delayed replacement of these cells in the basal layer, resulting in ulceration on the mucosal surfaces (Sonis, 2004). However, healthcare professionals now know that the pathogenesis of mucositis is multifactorial and more complex than simply epithelial damage (Lalla et al., 2008). Sonis (2004) described a five-stage model of evidence-based pathogenesis of mucositis (see Table 1). Inflammatory cells and pro-inflammatory cytokines, including cyclo-oxygenase-2, nuclear factor-kappa B, tumor necrosis factor-alpha, and interleukin-6, are upregulated in oral mucositis (Lalla et al., 2008). The identification of these pathways paves the way for understanding the pathogenesis of oral mucositis and provides potential therapeutic targets for the development of new therapies.

When patients are immunosuppressed, ulcerations are more likely to become infected and are potential entry sites for HSV. Knowing the pathophysiology of HSV is critical in caring for patients with lymphoma who develop oral mucositis when receiving chemotherapy.

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