Hemorrhagic Cystitis

Treatment with hyperbaric oxygen therapy in patients with acute lymphoblastic leukemia

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BACKGROUND: Hyperbaric oxygen therapy is a rare treatment modality for hemorrhagic cystitis (HC) following BK virus reactivation in the immunosuppressed population. Clinicians need to be aware of the etiology, preventive measures, complications, and various management techniques in HC while treating patients undergoing bone marrow transplantation.

OBJECTIVES: This study details the pathologic progression of HC in a patient with acute lymphoblastic leukemia harboring BK virus after cytotoxic induction chemotherapy and haploidentical marrow transplantation.

METHODS: A search of PubMed for literature published from 1973–2018 was conducted using keywords.

FINDINGS: Hyperbaric oxygen therapy in chemotherapy-induced and BK virus–associated HC is a viable management option in parallel with tapering of immunosuppressives, bladder irrigation, and IV resuscitation within the post-transplantation acute lymphoblastic leukemia population.

KEYWORDS
hemorrhagic cystitis; hyperbaric oxygen; BK virus; acute lymphoblastic leukemia

DIGITAL OBJECT IDENTIFIER
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HEMORRHAGIC CYSTITIS (HC) IS ASSOCIATED WITH BK VIRUS REACTIVATION in the immunosuppressed transplantation population after exposure to various cytotoxic therapies, most commonly the alkylating agents cyclophosphamide and ifosfamide. In addition, the incidence of HC is common with busulfan; rare with thiotepa, fludarabine, and chlorambucil; 20%–30% with doxorubicin; and 17% with cabazitaxel (Moy, Linder, Chao, & Grounder, 2018). The annual incidence post-transplantation ranges from 3%–24% and is associated with renal and hemorrhagic pathology (Gilis et al., 2014). HC is clinically defined as minimal or massive based on either the presence of microscopic hematuria (5–50 red blood cells per high power field) or the necessity of blood transfusion, respectively. For clinical diagnosis of HC, diffuse bladder inflammation, microscopic hematuria (red blood cell per high power field on urinalysis), or significant hematuria requiring red blood cell infusion is required. Massive blood loss with subsequent transfusion disrupts the normal coagulation, fibrinolysis, and platelet mechanism, sometimes leading to disseminated intravascular coagulation (DIC). The combination of malignancy, infection, and bleeding diathesis incites DIC at a higher rate than if a patient had any one cause alone (Russo, 2000). With massive bleeding, mortality from HC increases to about 2%–4%, and subsequent complications (hydronephrosis, anemia, and thrombocytopenia) generally require prolonged hospitalization (Watson & Notley, 1973). These extended institutional stays expose immunosuppressed patients to various opportunistic pathogens during a period of heightened risk. As hematopoietic transplantation rates increase, clinicians should understand these complex complications and their management.

The immunosuppressed are particularly vulnerable to HC because of their immune attenuation and direct urothelial contact with cyclophosphamide or ifosfamide (Arthur, Shah, Baust, Santos, & Saral, 1986) (see Figure 1). Urotoxic acrolein, a liver metabolite of both compounds, is excreted into the renal tubules. The bladder, acting as a storage vessel, has prolonged exposure to its inflammatory effects. Acrolein toxicity disrupts the normal transitional epithelium to induce polyuria, dysuria, urgency, and suprapubic discomfort, in addition to micro- and macroscopic blood loss. Catastrophic events, such as bladder rupture, can occur with chronic inflammation (Imataki, Uchida,