Background: Adult patients with cancer receiving antineoplastic, targeted, and other immunosuppressive therapies are at risk for severe side effects. Studies link posterior reversible encephalopathy syndrome (PRES) with immunosuppressants used for patients undergoing transplantation, as well as select tyrosine kinase inhibitors (TKIs) and other targeted therapies used in patients with cancer. PRES is a reversible condition with early recognition and management; however, permanent neurologic toxicities have been reported.

Objectives: This article aims to educate oncology nurses on signs, symptoms, and management of PRES in patients receiving TKIs.

Methods: The literature was reviewed to develop an educational session about causes, manifestations, pathophysiology, and management of PRES. Using a case study and flipped classroom model, staff participated in an online lecture and concept engagement exercise. Education for nurses included frequent neurologic and mental status assessments, blood pressure monitoring with mean arterial blood pressure goal, and seizure precautions. Nursing knowledge was evaluated with pre- and post-testing.

Findings: Evaluation revealed improved knowledge in recognizing and managing patients with PRES related to TKIs. The flipped classroom approach was perceived as a valuable tool for busy staff nurses.

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Key words: biotherapy; targeted therapies; leukemia; lymphoma; hematologic; management issues; neurotoxicity; oral therapies

Digital Object Identifier: 10.1188/16.CJON.305-308

Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic disorder with specific neuroimaging findings (Bartynski, 2008). Hinchey et al. (1996) first described PRES as a clinicoradiologic entity based on 15 observed cases. The pathophysiologic etiology for PRES is unknown, but some hypotheses for its cause exist. The syndrome is usually diagnosed by its common symptoms and neuroimaging findings. It can be identified using computed tomography (CT) and magnetic resonance imaging (MRI) findings along with significant clinical changes (Legriel, Pico, & Azoulay, 2011).

Tyrosine kinase inhibitors (TKIs) are one of the known agents that can cause PRES (Neill, 2016). TKIs work in cancer therapy by binding to the adenosine triphosphate binding site on malignant cells, where they inhibit angiogenesis and proliferation (Yu, Steeghs, Nijenhuis, Schellens, & Huitema, 2014). According to Yu et al. (2014), TKIs are used to treat several disorders, such as acute lymphoblastic leukemia, chronic myelogenous leukemia, renal cell