## Toxic Leukoencephalopathy: A Review and Report of Two Chemotherapy-Related Cases

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oxic leukoencephalopathy is a rare disorder that is characterized by edema of cerebral white matter (Filley & Kleinschmidt-DeMasters, 2001). Because this syndrome alters neurobehavioral function, patients may present in a confused state, which can progress quickly to irreversible dementia, coma, or death, depending on its severity (Cossaart, SantaCruz, Preston, Johnson, & Skikne, 2003; Filley, 1999; Filley & Kleinschmidt-DeMasters). Caused by toxins, such as chemotherapy agents, the prevalence of this disorder is unknown; however, it has been reported increasingly in the literature. Two cases of chemotherapy-related leukoencephalopathy are described in this article, along with a review of the current literature.

Leukoencephalopathy syndrome is a rare disorder that results from structural alterations of cerebral white matter, is characterized by cerebral edema, and can occur in patients of any age. Cranial irradiation and certain chemotherapy agents, especially those used in high-dose protocols, are causal agents. The prevalence of toxic leukoencephalopathy is unknown; however, this syndrome has been reported increasingly in the literature in patients who develop neurobehavioral changes following exposure to various toxins. Diagnosis must confirm exposure to a toxin and the presence of neurobehavioral deficits and neuroradiologic abnormalities. In most reported cases, clinical symptoms are reversible after the offending toxin is withdrawn. This article describes two cases of chemotherapy-related leukoencephalopathy and reviews the nursing care of patients experiencing this syn-

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## **Case Reports**

Ms. C, who is 66 years old, developed leukoencephalopathy following high-dose melphalan and stem cell rescue for multiple myeloma. Ms. W, a 50-year-old with acute myeloid leukemia, developed the syndrome following cytarabine and daunorubicin chemotherapy and methylprednisolone administration for an erythema multiforme rash. In both cases, complete resolution of symptoms occurred, although case reports have shown the course progresses rapidly, resulting in death (Cain, Burton & Holcombe, 1998; Cossaart et al., 2003).

## Case Study 1

Ms. C was admitted to an acute private hospital in Australia on June 10, 2002, for high-dose chemotherapy and an autologous stem cell transplant to treat her multiple myeloma. On the third day of admission in preparation for the transplant, she received melphalan 200 mg/m² (380 mg) as the conditioning regimen. Twenty-four hours after melphalan administration, Ms. C was reinfused with 7.08 x 106 CD34 cells. She subsequently experienced nausea and vomiting, fluid retention with a weight gain greater than 1 kg that required 40 mg of furosemide

via IV on days 5–7 of her admission, and moderate to severe mucositis and esophagitis that necessitated a morphine infusion on day 10 of her admission. Neutropenic fever occurred on day 7; as a result, ticarcillin and clavulanic acid, gentamicin, and vancomycin were initiated.

On day 13 of her admission, Ms. C became increasingly confused. Her blood pressure (BP) was 140/90 mm/Hg, and her electrolytes were within normal limits, except for an increased level of creatinine from 0.81 mmol/l to 1.34 mmol/l (normal range = 0.04-0.12 mmol/l). Her confusion persisted, and she became disoriented to time and place. No headaches were reported. A computed tomography (CT) scan of the head performed at this time was normal, and biochemical and hematologic parameters were within normal limits. On day 16, Ms. C re-

mained disoriented to time and place, was agitated, and developed hypertension (BP 196/90 mm/Hg). Further neurologic deterioration occurred: The patient was unresponsive to verbal commands, her eyes deviated to the right, and she developed cortical blindness (i.e., loss of sight because of a lesion in the cortical representation of vision, yet the fundus and pupillary reflexes are normal). On day 17, a lumbar puncture yielded

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