

# Identifying and Managing Complications of Whole Brain Radiotherapy

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S.D. is an 85-year-old man initially diagnosed in 1996 with a cutaneous malignant melanoma on his scalp. His disease has slowly progressed during the past several years, requiring multiple surgeries, localized scalp and neck irradiation, chemotherapy, and systemic biologic agents, with overall good systemic disease control. Six months ago, S.D. showed signs of clinical progression of subcutaneous disease of the left neck and axillae. Repeat computed tomography scans revealed overall progression of existing subcutaneous and pulmonary disease, and new brain lesions were discovered. Magnetic resonance imaging (MRI) with contrast revealed diffuse innumerable supratentorial brain metastases measuring 2–5 mm. The largest lesion was located in the right parietal lobe and measured 1.1 x 1 x 0.9 cm with surrounding vasogenic edema. Based on the large quantity of diffusely scattered lesions, stereotactic radiosurgery and surgical resection were not appropriate options. A course of whole brain radiation therapy (WBRT), delivering 37.5 grey over three weeks was deemed the best approach to palliate S.D.'s brain metastases.

Complications that developed during the first four months after irradiation had a significant impact on S.D.'s physical and neurocognitive function. Those complex yet often predictable problems are sequelae of WBRT and the medications necessary to manage disease- and treatment-related effects. The long-lasting, debilitating effects of therapy have proven to be very challenging for S.D., his wife, and the multidisciplinary team caring for him.

## Patient Assessment

Prior to WBRT, S.D.'s performance status was 90 on a Karnofsky Performance Scale (KPS) and 28 of 30 on a Mini-Mental Status Examination (MMSE). His primary complaints were those of National Cancer Institute Common Toxicity Criteria for Adverse Events grade I fatigue, 3 of 10 left neck subcutaneous tumor pain, memory deficits, and generalized weakness. Upon diagnosis of the new brain metastases, a glucocorticoid steroid, dexamethasone 4 mg four times a day, and anti-epileptic levetiracetam 500 mg twice a day were started as a prophylaxis for disease and radiation-induced symptoms.

After completing WBRT, S.D. developed a number of anticipated complications and symptoms, including grade II–III fatigue, steroid myopathy, persistent generalized weakness, gait imbalance, anorexia, forgetfulness, mild headaches, and steroid-induced diabetes. However, he did not experience nausea, vomiting, or seizure activity, all of which commonly occur with the presence of brain metastases (Lovely, 2004).

Repeat MRIs two and three months after WBRT showed stable disease, but a mild increase in cerebral edema was noted and managed with an escalation in dexamethasone dosage. Attempts to taper and discontinue daily oral dexamethasone dosing had been problematic. A reduction

of less than 4 mg daily exacerbated S.D.'s symptoms related to cerebral edema.

## Etiology of the Problem

S.D. is one in an estimated 170,000 people annually living in the United States with cancer who have developed brain metastases from their primary tumors (Khosla, 2008; Majer & Samlowski, 2007). Melanoma ranks third in occurrence after lung cancer and breast cancer for the primary site of brain metastases (Majer & Samlowski). Patients with pulmonary metastases have a higher risk of metastatic cells traveling via arterial circulation to the brain. The location of metastatic deposits tends to correlate with the path of blood flow (Eichler & Loeffler, 2007; Khosla). Metastatic lesions most commonly occur in the cerebrum (supratentorium) with an 80%–85% incidence, followed by the cerebellum (infratentorium) with a 10%–15% incidence, and the brain stem with a 3%–5% incidence (Khosla; Lovely, 2004) (see Figure 1). Multiple sites of metastases commonly are discovered within the brain versus a solitary metastatic lesion from melanoma. A solitary metastatic lesion may be treated with local irradiation or surgery; however, new tumors often develop, particularly if systemic disease is controlled poorly (Khosla).

The pathophysiologic changes associated with WBRT alter the endothelium of the vessels and cause demyelination

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