

Seize the Moment to Learn About Epilepsy in People With Cancer

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Seizures have been recognized for more than a century as a symptom of primary and secondary intracerebral tumors (Beaumont & Whittle, 2000). Seizures can occur at any point during the course of the disease and even have been reported to predate the diagnosis of cancer by many years (Beaumont & Whittle; Goldring, Rich, & Picker, 1986). When seizures occur in patients with cancer, the disorder often is referred to as “tumor-associated epilepsy,” a subtype of the classic condition of epilepsy.

Epilepsy or a seizure disorder can be defined as an intermittent derangement of the nervous system, presumably because of a sudden, excessive, disorderly discharge of cerebral neurons (Adams & Victor, 1993). Seizures occur in patients with cancer as a direct effect of the cancer on the nervous system or as a result of the metabolic impact of the cancer or cancer treatment (Gilbert & Armstrong, 1995). The consequences of a seizure occurring in patients with cancer can include worsening of neurologic dysfunction, slow recovery of a neurologic deficit, or even death (Cairncross, 1983). Therefore, a thorough search for the cause of a seizure, as well as appropriate medical management, is imperative in patients with cancer. The purpose of this article is to define and describe the etiology, pathophysiologic basis, and management of seizures in patients with cancer.

Etiology of Seizures

Seizures can occur at any point in the course of illness and are often the first sign of neurologic dysfunction in patients with

Seizures are common neurologic complications of cancer and can occur at any point in the disease trajectory. Despite this, the exact pathophysiologic basis of seizures related to cancer is not known. The etiology of seizures is thought to be multifactorial, including the presence of tumors within the cranial cavity, metabolic derangements, and the direct effect of medications on the central nervous system. Seizure management often employs anticonvulsant medications and interventions to promote patient safety. Oncology nurses must be aware of the potential seriousness of this complication and implement appropriate strategies to assist patients in maintaining safety and quality of life.

Key Words: seizures, status epilepticus, anticonvulsants

cancer (Cairncross & Posner, 1981). Seizures can result from a structural lesion within the brain (e.g., direct tumor involvement, vascular events, central nervous system [CNS] infections) or as a consequence of toxins or metabolic derangements.

The occurrence of seizures is influenced by the tumor type and the location of the tumor within the CNS. Tumors involving the brain parenchyma, dura, or spinal fluid may be associated with seizures. Seizures also are reported in patients with extracerebral, intracranial tumors such as meningiomas. Low-grade primary tumors (e.g., astrocytomas, oligodendroglioma, oligoastrocytoma, ganglioglioma, pleomorphic xanthoastrocytoma) are associated with a higher incidence of seizures than malignant tumors, such as glioblastoma multiforme (GBM) and anaplastic astrocytoma (Beaumont & Whittle, 2000; Cascino, 1990). Patients with oligodendrogliomas have the highest incidence of seizures (89%–90%), whereas patients with GBM have a 31%–40% incidence (Beaumont & Whittle). Meningiomas, the

most common extracerebral tumors, are associated with a 29%–41% seizure incidence (Beaumont & Whittle). Patients with metastatic tumors have a 35% risk of seizures, and nearly 25% occur as the first sign of neurologic dysfunction in patients with a preexisting diagnosis of cancer (Beaumont & Whittle). CNS tumor location also influences the likelihood of seizure occurrence; tumors located within the frontal, parietal, and temporal cortex are associated with a higher incidence of seizures than those in the occipital lobe. Tumors of the thalamus, basal ganglia, and cerebellum are not known to

produce seizures.

CNS ischemic or hemorrhagic vascular events can provoke seizures. These seizures can result from tumor-induced hypercoagulable states or as a consequence of stroke associated with endocarditis (Collins, Al-Mondhry, Chernik, & Posner, 1975; Rosen & Armstrong, 1973). CNS infections, especially in immunocompromised hosts, include bacterial or fungal meningitis, brain abscess, or viral encephalitis and are known to trigger seizures.

Toxic or metabolic abnormalities can cause seizures with or without a coexisting structural lesion. Metabolic factors include hepatic and renal failure, electrolyte abnormalities,

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