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Short Communication

The Effects of Chemotherapy on the Central Nervous System

Richard Ambinder*

Departments of Medicine and Oncology, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

ABSTRACT

The dosage of the drug is consistently related to nervous system impairment. After brain radiation therapy or chemotherapy medications infused into the cerebrospinal fluid, damage to the central nervous system is more likely. The CNS can be affected by cancers that start in the brain or spinal cord or have spread to the brain or spinal cord. Chemotherapy has a negative impact on cognitive performance both immediately and chronically, although the underlying processes are unknown. Short-term chemotherapy produces not just acute destruction to progenitor cells, but also delayed damage to myelin, according to a recent study.

Keywords: Chemotherapy; Central nervous system; Children

DESCRIPTION

Many cancer patients are treated with a variety of treatments, such as radiation and chemotherapy. The mechanisms underpinning toxic adverse effects of systemic chemotherapy on the Central Nervous System (CNS) have not been extensively defined, in contrast to the well-documented toxic effects of brain radiation, which have been recognized for a long time [1]. Since both systemic chemotherapy and brain radiation can cause severe neurotoxicity, patients who receive both treatments are more likely to experience neurotoxic side effects. Children treated with chemotherapy and radiation for brain tumors and other types of cancer have long been known to experience cognitive impairment [2]. Practically all types of chemotherapeutic drugs have been associated to neurotoxic adverse effects. Despite a vast number of clinical investigations and case reports confirming both acute and long-term neurotoxicity as a result of anticancer treatment, however little understood about the molecular mechanisms driving such nervous system damage. Chemotherapy-related CNS issues can be the result of the drug's direct harmful effects on nervous system cells, or they can be produced indirectly by metabolic abnormalities, inflammatory processes, or vascular side effects [3].

Patients receiving localized or systemic chemotherapy are at risk of acquiring a wide range of neurotoxic side effects, and long-term neurological issues are frequently related with survival. Neurotoxic syndromes can manifest as acute, sub-acute, or delayed consequences, and they can occur years after treatment has ended. Cognitive impairment, white matter disease, cerebral atrophy, and dementia are examples of delayed neurologic consequences. A number of chemotherapeutic medications have been linked to neurotoxicity, including alkylating agents (Eg: carmustine and cisplatin), antimetabolites (Eg: cytosine arabinoside, 5-fluorouracil, and methotrexate), mitotic inhibitors (Eg: vincristine), and antihormonal agents (Eg: tamoxifen) [4]. For instance, methotrexate and carmustine are associated with a high rate of neurotoxicity, which can be severe and progressive, especially if the medicine is given following radiation therapy. Both drugs have been linked to a well-known leukoencephalopathy syndrome, especially when given at high doses, intrathecally, or in combination with cranial irradiation [5].

Several cytotoxic drugs, including as cyclophosphamide, cisplatin, ifosfamide, and thiotepa, were linked to considerable and dose-dependent neurotoxicity in multiple brain areas, including the cortex, basal ganglia, and hippocampus, according to other research. These investigations, on the other hand, didn't reveal anything about the lineage-specific effects of chemotherapy on the brain. The oligodendroglial lineage has been thought to be particularly sensitive to alkylating agents, which is consistent with the clinical observation that oligodendrogliomas and astrocytomas often respond differently to chemotherapy [6]. It was discovered that dividing neural progenitor cells, which are the direct ancestors of all differentiated cell types of the CNS and oligodendrocytes, are the

Correspondence to: Richard Ambinder, Departments of Medicine and Oncology, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA, E-mail: rambind1@jhmi.edu

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most vulnerable cell populations to the effects of multiple chemotherapeutic agents, using a detailed lineage-based approach to test the effects of commonly used chemotherapeutic agents on mature and immature cell types of the nervous system. Nondividing oligodendrocytes were just as vulnerable as oligodendrocyte precursor cells, indicating that vulnerability was not limited to dividing cells. In experimental research, neurotoxicity has also been shown after systemic administration of thiotepa and methotrexate. Both medications were found to suppress hippocampus cell proliferation in a dose-dependent manner. Furthermore, as a functional consequence of chemotherapy-related damage, methotrexate has been demonstrated to decrease cognitive ability [7].

CONCLUSION

There is also considerable evidence that cancer treatment, like as chemotherapy and radiation, can affect progenitor cells, oligodendrocytes, white matter tracts, gliogenesis, and neurogenesis directly. Damage to brain progenitor cells has been proposed as a plausible explanation for delayed toxicities like cognitive impairment, cerebral atrophies, and white matter damage. However, clinical and experimental evidence suggests that other factors are likely to have a role in determining the likelihood and severity of neurotoxicity. In order to avoid unnecessary toxicities, future studies will need to uncover aspects and mechanisms that influence CNS toxicity, as well as develop and improve specific therapy.

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