This problem was FIRST identified by people with cancer who had undergone conventional treatments. They were not believed in the beginning. It took years before this problem/harm was recognized by clinicians and studies by researchers. Here are some articles that may be of interest.

Cancer as a Risk Factor for Long-Term Cognitive Deficits and Dementia

Previous studies have shown that cancer survivors frequently experience short-term cognitive deficits, but it is unknown how long these deficits last or whether they worsen over time.

Using a co-twin control design, the cognitive function of 702 cancer survivors aged 65 years and older was compared with that of their cancer-free twins.

Dementia rates were also compared in 486 of the twin pairs discordant for cancer. Cancer survivors overall, as well as individuals who had survived cancer for 5 or more years before cognitive testing, were more likely than their co-twins to have cognitive dysfunction (odds ratio [OR] = 2.10, 95% confidence interval [CI] = 1.36 to 3.24; P<.001 and="" or="2.71," ci="1.47" to="" p="" respectively="">
Cancer survivors were also twice as likely to be diagnosed with dementia as their co-twins, but this odds ratio did not reach statistical significance (OR = 2.0, 95% CI = 0.86 to 4.67; P = .10).

These results suggest that cancer patients are at increased risk for long-term cognitive dysfunction compared with individuals who have never had cancer, even after controlling for the influence of genetic factors and rearing environment.

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Chemobrain: A review on mechanistic insight, targets and treatments

Abstract

Chemo-brain refers to the thinking and memory problems that occur in cancer patients during and after chemotherapy. It is also known as cognitive dysfunction or chemo-fog. Risk factors include brain malignancies, either primary or metastatic, radiotherapy and chemotherapy, either systemic or brain targeted. There are various mechanisms by which chemo-brain occurs in patients post-chemotherapy, including inflammation of neurons, stress due to free radical generation, and alterations in normal neuronal cell process due to biochemical changes. While chemotherapy drugs that are non-brain targeted, usually fail to cross the blood-brain barrier (BBB), this is not the case for inflammatory cytokines that are released, which easily cross the BBB. These inflammatory neurotoxic agents may represent the primary mediators of chemobrain and include the pro-inflammatory cytokines such as interleukins 1 and 6 and tumor necrosis factor. The pronounced rise in oxidative stress due to continuous chemotherapy also leads to a reduction in neurogenesis and gliogenesis, loss of spine and dendritic cells, and a reduction in neurotransmitter release. Based on recent research, potential agents to prevent and treat chemo brain have been identified, which include Lithium, Fluoxetine, Metformin, Rolipram, Astaxanthin, and microglial inhibitors. However, more defined animal models for cognitive dysfunction are required to study in detail the mechanisms involved in chemo-brain; furthermore, well-defined clinical trials are required to identify drug targets and their therapeutic significance. With these focused approaches, the future for improved therapies is promising.

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Dietary agents in mitigating chemotherapy-related cognitive impairment (chemobrain or chemofog): first review addressing the benefits, gaps, challenges and ways forward

Abstract

Chemobrain or chemofog is one of the important but less investigated side effects, where the cancer survivors treated with chemotherapy develop long-term cognitive impairments, affecting their quality of life. The biological mechanisms triggering the development of chemobrain are largely unknown. However, a literature study suggests the generation of free radicals, oxidative stress, inflammatory cytokines, epigenetic chromatin remodeling, decreased neurogenesis, secretion of brain-derived neurotropic factor (BDNF), dendritic branching, and neurotransmitter release to be the cumulative contributions to the ailment. Unfortunately, there is no means to prevent/mitigate the development and intensity of chemobrain. **Given the lack of effective prevention strategies or treatments, preclinical studies have been underway to ascertain the usefulness of natural products in mitigating chemobrain in the recent past. Natural products used in diets have been shown to provide beneficial effects by inhibition of free radicals, oxidative stress, inflammatory processes, and/or concomitant upregulation of various cell survival proteins. For the first time, this review focuses on the published effects of astaxanthin, omega-3 fatty acids, ginsenoside, cotinine, resveratrol, polydatin, catechin, rutin, naringin, curcumin, dehydrozingerone, berberine, C-phycocyanin, the higher fungi *Cordyceps militaris*, thyme (*Thymus vulgaris*) and polyherbal formulation Mulmina™ in mitigating cognitive impairments in preclinical models of study, and also addresses their potential neuro-therapeutic mechanisms and applications in preventing/ameliorating chemobrain**.

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