

Leukemia Developing after ¹³¹I Treatment for Thyroid Cancer

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Radioactive iodine has been used to treat hyperthyroidism since 1940 [1]. Radioiodine (¹³¹I) is used in the treatment of thyroid cancer since 1946 in order to eliminate residual thyroid tissue following total or near total thyroidectomy for papillary or follicular carcinoma larger than 1 to 1.5 cm and treat to metastatic disease [2-5]. Acute risks associated with RAI therapy include nausea and vomiting, ageusia (loss of taste), salivary gland swelling and pain. Longer-term complications include recurrent sialoadenitis associated with xerostomia, mouth pain, dental caries, pulmonary fibrosis, nasolacrimal outflow obstruction, effect on fertility and second primary malignancies [6,7]. Some authors feel that the risk of a secondary tumor associated with ¹³¹I therapy is low and lacks clinical impact [8,9]. The relative risk approximates 1.21 to 1.9 per 10,000 patient years compared to the general population and is greatest for hematologic malignancies [10]. Leukemia is rare complication or late effect of exposure to RAI therapy and was first reported in 1955, almost all the cases have occurred after cumulative dosage of more than 800 mCi, in patients more than 50 years of age and with intervals between dosage of RAI less than 12 months. Although cases of patients developing leukaemia after low-dose radioactive iodine are reported, the link with the treatment is still a matter of debate [2,3,10]. Radioactive iodine therapy has been the treatment of choice for toxic nodular goiter, Grave disease, and thyroid cancer. The relative risk of second primary malignancy in thyroid cancer patients is higher in those treated with RAI than those without [6,11]. Therapy-related acute myeloid leukemia (t-AML) is well described after chemotherapy or radiotherapy for diverse malignancies. Radioisotope therapy is also recognized as a less-common cause of t-AML and occurs in less than 2% of thyroid cancer patients and is associated with a poor therapeutic response and prognosis [12,13]. Ionizing radiation like radioiodine-induced sub-lethal damage to bone marrow, which leads to chromosomal aberrations and oncogene activation causing hematological malignancy, are postulated [6,11]. A strict hematological follow-up is warranted in such patients, for early detection of myelodysplastic syndromes, leukemia's, or other hematological disorders. This type of complications stresses the need for specific research programs focusing on the risk factors that influence the occurrence of secondary acute leukemia (SAL) in cancer survivors. However, the possibility that the emergence of SAL can be connected just as much to environmental factors as to thyroid cancer cannot be excluded and larger scale studies may help us identify them.

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