Ischemic Stroke After Radioactive Iodine Therapy In Graves Disease

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Disclosure: T. Rave: None. J. Giunta: None.

Hyperthyroidism is defined as excess thyroid hormone, most commonly due to Graves Disease. Thioridines are first line therapy, however more definitive options are radioactive iodine (RAI) or thyroidectomy. RAI enters thyroid follicular cells and destroys the thyroid tissue with beta ray emissions. Patients with Graves disease, toxic multinodular goiter, and thyroid cancer can benefit from RAI, but it is contraindicated in pregnancy, breast feeding, and severely uncontrolled thyrotoxicosis. Early side effects are pancytopenia, thyrotoxicosis, and later complications are bone marrow suppression and cancer. There are several reports of cerebral vascular accidents (CVA) after RAI therapy, however, there is limited research demonstrating whether CVA is a direct consequence of RAI or secondary to thyrotoxicosis. This case describes a possible RAI-induced CVA. A 60-year-old female with a past medical history of type 2 Diabetes Mellitus, hypertension, obesity, and Graves disease presented with altered mental status and gait instability for 1 day. She received RAI therapy 1 day prior to symptom onset for uncontrolled Graves disease. She was previously on Methimazole and PTU, however, both were discontinued due to an intolerable rash. As a result, she was maintained on a beta blocker prior to RAI. On presentation, she was afebrile, normotensive with a heart rate of 79. She was oriented only to person and place and had an unbalanced gait. Her Burch-Wartofsky score was 30, which was concerning for thyrotoxicosis. Her TSH was undetectable and EKG showed normal sinus rhythm. She was placed on atenolol, PTU and hydrocortisone. Her CT head was negative for acute disease, however, her MRI brain diagnosed an acute ischemic stroke. During admission, she had episodes of atrial fibrillation with rapid ventricular response. This patient presented with thyrotoxicosis after RAI therapy complicated by a CVA. It is important to note that on presentation she was reliably taking a beta blocker outpatient and her EKG was normal sinus rhythm making atrial fibrillation a less likely culprit of her CVA. Research has found that hyperthyroidism alone can lead to a prothrombotic state, however, additional literature reports that both hyperthyroid and euthyroid patients have an increased risk of CVA following RAI. Whether a CVA is secondary to thyrotoxicosis or a direct effect of RAI, this potentially life-threatening complication should be discussed with patients prior to treatment.

Presentation: Friday, June 16, 2023