IODINE-INDUCED HYPERTHYROIDISM: DO YOU MIND?

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Abstract: Iodine can be obtained by consumption of the foods that include it or to which it is added. Iodine-induced hyperthyroidism, also known as Jod-Basedow Syndrome, usually occurs in the cases of an underlying thyroid disease, such as autoimmune thyroid disease, latent Graves disease, non toxic diffuse or nodular goiter or previous thyroid surgery. Iodine-induced hyperthyroidism is frequently self-limited with a period of 1-18 months after the discontinuance of iodine supply/replation. The prognosis is frequently favorable, the majority returns to the baseline hormonal status. The long term outcomes of Jod-Basedow effects remain unknown due to the scarcity of phenomenon. Nonetheless, it is remarkable that treatment, devoted to the underlying thyroid diseases, should be addressed after resolution of the acute episode. Furthermore, an interprofessional health care team must serve and officiate not only to treat iodine-induced hyperthyroidism, but also to concern themselves actively in prophylaxis.

Key words: Iodine; Hyperthyroidism; Jod-Basedow; Thyroid; Endocrine Surgery; Thyroidology.

IODINE-INDUCED HYPERTHYROIDISM

Iodine can be obtained by consumption of the foods that involve it or to which it is annexed. Dietary iodine is absorbed from stomach and duodenum in the rate of > 90% as iodide and rapidly distributed in the extracellular fluid, transported to the thyroid gland by sodium/iodide symporter with a gradient of 25-50 times of plasma. The recommended minimum daily intake of iodine is 150 μg for nonpregnant adults, > 12 years; 220 to 250 μg for pregnant women, and 90 μg to 120 μg for children, 0-59 months and 6-12 years, respectively (1).

The prevalence of hyperthyroidism that has been reported subsequent to the iodine administration is 1-20%, while the higher frequency, 10-20%, appears in the individuals with nodular goiter living in the areas of iodine deficiency (2, 3). Iodine-induced hyperthyroidism, also known as Jod-Basedow Syndrome, scarcely occurs in cases in the absence of an underlying thyroid disease like autoimmune thyroid disease, previous thyroid surgery, latent Graves disease, and non-toxic diffuse or nodular goiter (4-8). The autonomy of underlying areas within the thyroid gland leads to thyrotoxicosis and the autonomous areas generate the thyroid hormones independently of normal regulatory mechanisms as exogenous iodine supply is augmented usually by iodinated contrast media used in conjunction with computed tomography scans, angiography, and various other imaging studies, or by intake of iodinated antiseptic solutions, and oral supplements (5, 9). A subclinical hyperthyroidism before iodine supply may exist in such cases (3).

In general, iodine-induced hyperthyroidism is self-limited with a period of 1-18 months after cessation of iodine administration. The preliminary treatment modality of iodine-induced hyperthyroidism is i) discontinuation of iodine administration, ii) abstaining from further exposure and iii) administration of a beta-adrenergic antagonists, such as atenolol 25-50 mg/day, as an initial dose, to diminish the symptoms of hyperthyroidism (10). Administration of a thionamide may expedite the recovery (11). Methimazole, possessing long duration of action, allowing for once-daily do-
sing, quicker efficacy and lower incidence of side effects, is recommended while the relevant symptoms are severe or prolonged (> 1 month) as starting dose of 10-20 mg/day. Corticosteroid therapy is suggested to accelerate the return of thyroid hormones to their normal ranges. In case, anti-thyroid drugs fail to attenuate thyroid hormone production, providers may regard to prescribe lithium, owing to its inhibitory impacts on the thyroid gland (5).

The prognosis is frequently affirmative, the majority of the cases returns to the baseline thyroid status. The complications of iodine-induced hyperthyroidism are consist permanent hyperthyroidism, thyroid storm, and atrial fibrillation (5). The longterm outcomes of iodine-induced hyperthyroidism, Jod-Basedow Effect, remain unknown because the phenomenon is rare. However, appropriate treatment leads to good outcomes in Thyroidology. Nevertheless, some cases may require the longterm treatment with antithyroid drugs and beta-blockers. Against all odds it is noteworthy that treatment of the underlying thyroid diseases should be addressed after resolution of the acute episode of Jod-Basedow Syndrome. In addition, the interprofessional health care team must serve and officiate not only to treat iodine-induced hyperthyroidism but also to concern themselves, actively in the relevant prophylaxis (5).

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Authors’ contribution
IS, originating the idea, constituting the notion, intellectual planning, design, co-writing, editing, and the final approval, agreement to be accountable for all aspects of the work; DS, originating the idea, constituting the notion, intellectual planning, design, and co-writing, editing, supervision, and the final approval, agreement to be accountable for all aspects of the work; AP, design, co-writing, editing, and final approval, agreement to be accountable for all aspects of the work.

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