IODINE-INDUCED HYPERTHYROIDISM: DO YOU MIND?

Sengul Ilker,^{1,2} Sengul Demet,³ Pelikán Anton^{4,5}

¹ Division of Endocrine Surgery, Giresun University Faculty of Medicine, Giresun, Turkey

² Department of General Surgery, Giresun University Faculty of Medicine, Giresun, Turkey

³ Department of Pathology, Giresun University Faculty of Medicine, Giresun, Turkey

⁴ Department of Health Care Sciences, Faculty of Humanities,

Univerzita Tomáše Bati ve Zlině, Zlín, Czech Republic

⁵ Department of General Surgery, Faculty of Medicine,
Ostravska Univerzita V Ostrave, Ostrava, Czech Republic

Primljen/Received 10. 07. 2020. god.

Prihvaćen/Accepted 30. 07. 2020. god.

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 rise 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/repletion. 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; 290 µg for lactating 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).

Acknowledgements

We thank all of the article participants.

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.

Conflict of Interests: The authors declare that there are no conflicts of interest related to this article.

Funding: None

Licensing

This work is licensed under a Creative Commons Attribution 4.0 International (CC BY 4.0) License.

Sažetak

JODOM-IZAZVAN HIPERTIREOIDIZAM: DA LI STE?

Sengul Ilker,^{1,2} Sengul Demet,³ Pelikán Anton^{4,5}

¹ Division of Endocrine Surgery, Giresun University Faculty of Medicine, Giresun, Turkey

² Department of General Surgery, Giresun University Faculty of Medicine, Giresun, Turkey

³ Department of Pathology, Giresun University Faculty of Medicine, Giresun, Turkey

⁴ Department of Health Care Sciences, Faculty of Humanities, Univerzita Tomáše Bati ve Zlině, Zlín, Czech Republic

⁵ Department of General Surgery, Faculty of Medicine, Ostravska Univerzita V Ostrave, Ostrava, Czech Republic

Jod se može uneti hranom koja ga sadrži ili kojoj je dodat. Jodom izazvan hipertireoidizam, također poznat i kao Jod-Basedow Sindrom, obično se javlja u slučajevima već prisutne bolesti štitaste žlezde, kao što su autoimuna bolest štitaste žlezde, latentna Graves-ova bolest, netoksična difuzna ili nodularna gušavost ili prethodna operacija štitaste žlezde. Jodom izazvan hipertireoidizam je često samolimitirajući tokom perioda 1-18 meseci nakon prekida unosa joda. Prognoza je često povoljna, veći-

na se vrati na početni hormonalni status. Dugoročni ishodi Jod-Basedow Sindroma ostaju nepoznati zbog retkosti i ograničenosti fenomena. Bez obzira na to, bitno je da lečenje postojeće bolesti štitaste žlezde bi trebalo nastaviti nakon akutne epizode. Multidisciplinarni tim medicinskih radnika mora ne samo lečiti jodom izazvan hipertireoidizam, već i aktivno se baviti profilaksom.

Ključne reči: Jod, Hipertireoidizam, Jod-Basedow, Štitasta žlezda, Endokrina hirurgija, Štitnjača.

REFERENCES

1. WHO, UNICEF, ICCIDD. Assessment of iodine deficiency disorders and monitoring their elimination: a guide for programme managers, third edition (updated 1st September 2008). Geneva, World Health Organization, 2001. Available at: www.who.int/nutrition/publications/micronutrients/iodine_deficiency/9789241595827/en/. Accessed June 10, 2020.

2. Martins MC, Lima N, Knobel M, Medeiros-Neto G. Natural course of iodine-induced thyrotoxicosis (Jod-basedow) in endemic goiter area: a 5 year follow-up. J Endocrinol Invest. 1989; 12(4): 239-44.

3. Roti E, Uberti ED. Iodine excess and hyperthyroidism. Thyroid. 2001; 11(5): 493-500.

4. Skare S, Frey HM. Iodine induced thyrotoxicosis in apparently normal thyroid glands. Acta Endocrinol (Copenh). 1980; 94(3): 332-6.

5. Rose HR, Zulfiqar H. Jod Basedow Syndrome. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing, 2020.
6. Al-Yaarubi S, Farhan H, Al-Futaisi A, Al-Qassabi S, Al-Rasadi K, Al-Riyami S, et al. Accuracy of ultrasound-guided fine-needle aspiration cytology for diagnosis of carcinoma in patients with multinodular goiter. *Indian J Endocrinol Metab.* 2011; 15(Suppl 2): S132-5.
7. Sengul D, Sengul I. Is there any link between a kind of thyrocyte dysfunction, hypothyroidism, and inflammatory hematologic parameters in the cases having the benign thyroid nodules? A 5-year single-centre experience. *Sanamed.* 2018; 13(1): 35-40.
8. Sengul D, Sengul I, Van Slycke S. Risk stratification of the thyroid nodule with Bethesda indeterminate cytology, category III, IV, V on the one surgeon-performed US-guided fine-needle aspiration with 27-gauge needle, verified by histopathology of thyroidectomy: The additional value of one surgeon-performed elastography. *Acta Chir Belg.* 2019; 119(1): 38-46.
9. Stanbury JB, Ermans AE, Bourdoux P, Todd C, Oken E, Tonglet R, et al. Iodine-induced hyperthyroidism: occurrence and epidemiology. *Thyroid.* 1998; 8(1): 83-100.
10. Bahn Chair RS, Burch HB, Cooper DS, Garber JR, Greenlee MC, Klein I, et al. Hyperthyroidism and other causes of thyrotoxicosis: management guidelines of the American Thyroid Association and American Association of Clinical Endocrinologists. *Thyroid.* 2011; 21(6): 593-646.
11. Burman KD, Wartofsky L. Iodine effects on the thyroid gland: biochemical and clinical aspects. *Rev Endocr Metab Disord.* 2000; 1(1-2): 19-25.

Correspondence to/Autor za korespondenciju

Demet Sengul, M.D.

Professor (Assoc) of Pathology

The Founder Chair, Department of Pathology

The Founder Chair, Scientific and Research Laboratories

Giresun University Faculty of Medicine

Gazipasa Compound, Gazi Avenue

TR28100 Giresun, Turkey

E-mail: demet.sengul.52@gmail.com